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TRANSACTIONS

OF THE

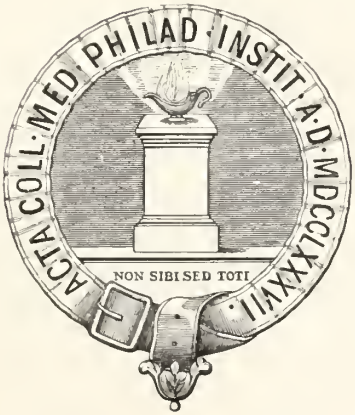
COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FIFTY-FOURTH



PHILADELPHIA
PRINTED FOR THE COLLEGE
1932

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1932, to December, 1932, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

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DORNAN, PRINTER

CONTENTS.

List of Presidents of the College vii
List of Officers and Standing Committees viii
List of Fellows of the College xi
List of Associate Fellows of the College lviii
Necrological List for 1932 lx
List of Prizes and Lectures lxi
Memoir of Francis X. Dercum, M.D. lxv
Memoir of Hobart Amory Hare, M.D. lxxii
Memoir of John Madison Taylor, M.D. lxxvi
Memoir of Charles Harrod Vinton, M.D. lxxxiii
Memoir of John B. Deaver, M.D. lxxxvii
Memoir of Charles Karsner Mills, M.D. xc

The President's Address. By Francis R. Packard, M.D. 1
Poliomyelitis. Mode of Infection and Means of Prevention. By Simon Flexner, M.D. 11
Prevention of Deformity in Infantile Paralysis. By DeForest P. Willard, M.D. 33
The Purposes of the Committee on the Costs of Medical Care. By Arthur C. Morgan, M.D. 37
Some Notes on Social Insurance and Health Work in Germany and Austria. By Henry Field Smyth, M.D., Dr.P.H. 41
The Plan of the British Medical Association for a General Medical Service for the Nation. By Samuel Bradbury, M.D. 44
The Curtis Clinic: Its Purpose and Its Possible Influence for the Advancement of Community Health. By Robert B. Nye, M.D. . . . 53
A Review of Some Recent Medical Literature Relating to Medical Economics, Costs of Illness, and Organization of Medical Practice. By Walter S. Cornell, M.D. 58
An Experiment in Psychoanalysis. By Earl D. Bond, M.D. . . . 69
The Increasing Incidence and Morbidity of Appendicitis. By John O. Bower, M.D. 76
Incidence of Tuberculosis in Infants and Pre-school Children and the Conditions Under Which They Become Infected. By J. C. Gittings, M.D. 84
The Incidence of Tuberculosis in Industrial Workers as Revealed by Fluoroscopic Examination. By Haynes Harold Fellows, M.D. . 90
The Occurrence and Progress of Tuberculous Lesions in School Children and Adults. By F. Maurice McPhedran, M.D. 98

Exogenous Tuberculous Infection of Adults. By Eugene L. Opie, M.D. 108
Cancer in Man. By Stanley P. Reimann 112
Prognosis in Carcinoma of the Stomach; Remote Results of Surgical Treatment to January, 1930. By Astley P. C. Ashhurst, M.D., and John W. Klopp, M.D. 116
Cancer: As a Problem of the Biology of Cell Division. By Frederick S. Hammett, M.D. 136
The Importance of the Differential Diagnosis of Tumors. By John H. Gibbon, M.D. 139
The Tumor Clinic. By Burton T. Simpson, M.D. 145
Cancer in Animals. By Herbert L. Ratcliffe, Sc.D. 152
Observations on the Epidemiology of Poliomyelitis. By John R. Paul, M.D., and J. D. Trask, M.D. 158
General Remarks on the Outbreak of Poliomyelitis in Philadelphia. By J. Norman Henry, M.D. 163
A Preliminary Report Concerning the Prophylactic Use of Parent's Blood During the Anterior Poliomyelitis Epidemic of 1932. By Howard C. Carpenter, M.D., and Joseph Stokes, Jr., M.D. 171
The Facial Expression of the Emotions with Special Reference to Violent Effort and Fatigue. The Nathan Lewis Hatfield Lecture, Lecture XI. By R. Tait McKenzie, M.D., LL.D., R.C.A. 174
A Summarization of the Advance in Our Knowledge of Some Features of Cancer. The Mütter Lecture. By H. Gideon Wells, M.D. . . 193
What Koch Meant to Tuberculosis. By Lawrason Brown, M.D. . . 211
Goethe and His Theory of Color. By Burton Chance, M.D. . . . 225

Proceedings of the Section on Ophthalmology

A Corneal Dystrophy. By J. Milton Griscom, M.D. 231
Posterior Lenticonus. By Warren S. Reese, M.D. 232
Sarcoma of the Choroid, Being a Clinico-pathological Distribution. By G. E. deSchweinitz, M.D., B. F. Baer, Jr., M.D., and Perce DeLong, M.D. 235
Histological Study of the Iris Inclusion Operation in the Eye of the Rabbit. By Edmund B. Spaeth, M.D. 237
Unilateral Neuroretinitis of Luetic Origin with Stellate Figure at the Macula. By A. G. Fewell, M.D. 238
Enucleation of the Eyeball, with Special Reference to the Use of a Tonsil Snare. By Luther C. Peter, M.D. 239
A Case of Bilateral Metastatic Carcinoma of the Choroid. By William Zentmayer, M.D. 241
Metastatic Sarcoma of the Choroid. By W. E. Fry, M.D. 243
Intraocular Sarcoma Which Filled the Orbit Before Medical Advice Was Sought. By H. Maxwell Langdon, M.D. 245
Sarcoma of the Choroid. By T. B. Holloway, M.D. 246
Diagnosis of Double Perforation of the Globe by Injection of Air into the Space of Tenon. By E. W. Spackman, M.D. 246

Allergic Investigations on Twenty-three Cases of Vernal Conjunctivitis. By Miss Ida Teller 249
Observations on Eighty-seven Cases of Vernal Conjunctivitis at the Wills Hospital. By Louis Lehrfeld, M.D. 250
Wound of the Globe by an Explosion of Shatter-proof Wind Shield (Presentation of Case). By H. Maxwell Langdon, M.D. . . . 255
Melanotic Sarcoma of the Choroid Occurring at the Optic Disk in an Eye with a Previous Detachment of the Retina. By H. Maxwell Langdon, M.D. 256
Microphthalmos with Coloboma of Iris, Choroid and Disk. By Hunter W. Scarlett, M.D. 257
Concerning Leber's Disease. By T. B. Holloway, M.D. 258
Syphilis in the Third Generation. By Joseph V. Klauder, M.D. . . 260
Amaurosis After Uterine Hemorrhage with Restoration of Vision Following Transfusion. By H. Maxwell Langdon, M.D., and George M. Laws, M.D. 262
Dr. Burton Alexander Randall as an Ophthalmologist. By Burton Chance, M.D. 266
Exhibition of a Case Presenting Disjunctive Ocular Movements. By Warren S. Reese, M.D. 268
A Case of Primary Sarcoma of the Iris. Removed by Iridectomy. By William Zentmayer, M.D. 269
A Case of Bullous Keratitis Successfully Treated by Iridectomy. By William Zentmayer, M.D. 270
A New Technique for the Preparation of Pathological Specimens for Microscopic Study. By Perce DeLong, M.D. 272
Report Upon 100 Intracapsular Cataract Extractions by the Knapp Method. By Leighton F. Appleman, M.D. 272
Fracture of Maxilla Through Left Optic Foramen. Reduction with a Kingsley Splint with Restoration of Vision. By H. Maxwell Langdon, M.D., and James E. Aiguier, D.D.S. 276
Extraction within the Capsule of the Crystalline Lens Containing a Foreign Body. With the Report of Three Cases. By Burton Chance, M.D. 277
Remarks Concerning Superficial Punctate Keratitis and the Associated Conjunctivitis. By G. E. deSchweinitz, M.D. 277
Metastatic Carcinoma of the Choroid. By Perce DeLong, M.D. . . 280
Sodium Carbonate (Monohydrated) in the Treatment of Vernal Conjunctivitis. By Louis Lehrfeld, M.D. 282

Proceedings of the Section on Otology and Laryngology

The Anatomy of the Paranasal Sinuses in Children. By J. Parsons Schaeffer, M.D. 285
Sinusitis in Children. By Warren B. Davis, M.D. 288
Lymphatic Drainage from the Maxillary Sinuses. By G. E. Pfahler, M.D. 293
Sinusitis and the Rôle It Plays in the Production of Asthma. By H. B. Wilmer, M.D. 294

Bronchial Asthma from the Rhinologic Standpoint. By Harry P. Schenck, M.D. 296
Intimate Notes on the Ear, Nose and Throat Clinic. By William Hewson, M.D. 300
Actinomycosis of the Tongue and Tonsil. By Robert F. Ridpath, M.D. 304
Studies on the Prophylaxis and Treatment of Septic Meningitis. By John A. Kolmer, M.D., and K. Amano, M.D. 305
Some Experience in the Treatment of Bilateral Abductor Paralysis. By W. B. Hoover, M.D. 308
The Otological Problem Presented by Suppuration of the Petrous Pyramid. By Samuel J. Kopetzky, M.D. 313
The Nature of the Static Labyrinth as Determined by Experiments in Amphibia. By William J. McNally, M.D. 318
Pulmonary Complications of Tonsillectomy, with Report of Three Cases. By James A. Babbitt, M.D. 320
Present-day Theories Concerning the Etiology and Pathology of Nasal Sinus Infections. By Benjamin D. Parish, M.D. 323
Some Studies of Pathologic Temporal Bones in Children. By Robert M. Wolff, M.D. 326
Joint Meeting with the Section on Otolaryngology of the New York Academy of Medicine. Postoperative Pulmonary Complications; Are They Preventable? (Lantern Demonstration.) By Chevalier Jackson, M.D. 330
Atresia of the Choanæ: Its Incidence, Types and Cause. (Lantern Demonstration.) By J. Parsons Schaeffer, M.D. 331
Discussion of Some of the Recent Developments in Regard to Otosclerosis, Cholesteatoma and the Static Labyrinth. By George M. Coates, M.D. 333
The Anatomy of the Tympanic Membrane (Demonstration with Manikin and Preparations). By O. V. Batson, M.D. 336
Preventriculosis (So-called Cardiospasm). Diagnosis and Treatment. (Moving Picture Film Demonstration.) By Chevalier L. Jackson, M.D. 338
Laryngofissure for Early Laryngeal Cancer. (Moving Picture Film Demonstration.) By Gabriel Tucker, M.D. 339
Operation on Abscess of the Temporosphenoidal Lobe. (Moving Picture Film Demonstration.) By Walter Roberts, M.D., and Henry Dintenfass, M.D. 340
Total Laryngectomy. One-stage Operation. (Lantern Demonstration of the Various Phases and Steps in this Operation.) By Fielding O. Lewis, M.D. 341
Proceedings of the Section on General Medicine 342
Proceedings of the Section on Public Health and Industrial Medicine 345
Proceedings of the Section on Medical History 346
Annual Report of the Library Committee 349

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES*
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
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1884	SAMUEL LEWIS†
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
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1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
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1919	WILLIAM J. TAYLOR
1922	THOMAS R. NEILSON
1925	HOBART A. HARE
1928	JOHN H. GIBBON
1931	FRANCIS R. PACKARD

* Died four months after his election.
† Resigned on account of ill-health.

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1932

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FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1932

* Non-resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., 4016 Pine St.

1912. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1926. Adler, Francis Heed, M.A., M.D., Assistant Surgeon, Wills Eye Hospital; Instructor in Ophthalmology and Physiology, University of Pennsylvania. 313 S. Seventeenth St.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Proctology in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1906. Allen, Francis Olcott, Jr., A.B., M.D. 2216 Walnut St.

1932. Allen, Frederick H., M.A., M.D., Director Child Guidance Clinic; Assistant Professor of Psychiatry, University of Pennsylvania Medical School. 1711 Fitzwater St.

1896. Allyn, Herman B., A.B., M.D., Consulting Physician to the Philadelphia General Hospital. 3910 Chestnut St.

1888. Anders, James M., M.D., Ph.D., LL.D., Sc.D., Emeritus Professor of Medicine in the Graduate School of Medicine, University of Pennsylvania; Officer de l'Instruction Publique, Chevalier Legion of Honor. 250 S. Seventeenth St.

ELECTED

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1930. Appel, Kenneth E., A.B., A.M., Ph.D., M.D., Assistant Professor of Psychiatry, Medical School, University of Pennsylvania; Chief of Out-Patient Department and Psychiatrist, Institute for Mental Hygiene, Pennsylvania Hospital; Chief of Neuro-Psychiatric Pay Clinic, Pennsylvania Hospital, Haverford, Pa.

1905. Appleman, Leighton F., M.D., Attending Surgeon to the Wills Eye Hospital; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School; Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Chief of Medical Service "B," Episcopal Hospital; Instructor in Medicine, Medical School and Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2116 Pine St.

‡1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. N. W. Cor. Twenty-second and Chestnut Sts.

1893. Ashton, Thomas G., M.D. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S., M.D., Professor of Research Medicine, University of Pennsylvania. John Herr Musser Department of Research Medicine, University of Pennsylvania.

1906. Babbitt, James A., A.B., A.M., M.D., Assistant Professor of Oto-laryngology, University of Pennsylvania; Associate Professor of Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist, Lankenau Hospital; Oto-laryngologist, Children's Hospital. 1912 Spruce St.

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Attending Surgeon to Wills Eye Hospital. 1927 Spruce St.

ELECTED

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital. 407 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1820 S. Rittenhouse Square.

1932. Barr, Everett S., M.D., Director, Chester County Hospital; Honorary Psychiatrist, Philadelphia General Hospital. West Chester, Pa.

1924. Bartle, Henry J., B. S., M.D., Clinical Assistant, Department for Disease of the Stomach and Bowels, Jefferson Medical College and Hospital. 1930 Chestnut St.

1931. Bates, William, M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Babies Hospital; Assistant Surgeon, Graduate Hospital, University of Pennsylvania. 2029 Pine St.

1932. Batson, Oscar V., M.D. 3502 Hamilton St.

1921. Bauer, Edward L., M.D., Professor of Children's Diseases, Jefferson Medical College; Pediatrist to the Jefferson and Memorial Hospitals and the Philadelphia Hospital for Contagious Diseases; Consulting Pediatrist, Germantown Hospital. 345 S. Nineteenth St.

*1883. Baum, Charles, A.M., M.D., Ph.D. 123 W. Main St., Middletown, Pa.

1922. Bazett, Henry Cuthbert, M.A., M.D., F.R.C.S., Professor of Physiology, University of Pennsylvania. Haverford, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P., Clinical Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-Patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 1919 Spruce St.

1930. Beardwood, Joseph T., A.B., M.D., Associate Physician, Chief of Diabetic Clinics, Presbyterian Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania, Physician in Chief, Department of Metabolic Diseases, Abington Memorial Hospital. 256 S. Twenty-first St.

1932. Behney, Charles A., A.B., M.D., Associate in Obstetrics and Gynecology, University of Pennsylvania; Obstetrician and Gynecologist, Bryn Mawr Hospital; Gynecologist to Chestnut

ELECTED

Hill and Radiologic Department, Philadelphia General Hospitals. 133 S. Thirty-sixth St.

1930. Belk, William Parks, A.B., M.D., Associate in Chemical Pathology, Graduate School of Medicine, University of Pennsylvania; Assistant Pathologist, Radiologic Clinic, Philadelphia General Hospital; Consulting Pathologist, Fitkin Memorial Hospital, Asbury Park, N. J., to Monmouth Memorial Hospital, Red Bank, N. J. and to the Bryn Mawr Hospital. Ardmore, Pa.

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy, Commanding Officer, U. S. Naval Medical School. 1869 Wyoming Ave., N.W., Washington, D. C.

1923. Bergey, David H., A.M., M.D., B.S., Dr. P. H., Professor of Hygiene and Bacteriology, University of Pennsylvania (retired); Director of Research in Biology, National Drug Company. 206 S. Fifty-third St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

1918. Billings, Arthur E., M.D., Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Hospital; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 2020 Spruce St.

1917. Birdsall, Joseph C., A.B., A.M., M.D., Associate Professor of Urology in the Graduate School of Medicine, and Urologist to the Hospital of the Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Presbyterian Hospital. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Professor of Obstetrics, Jefferson Medical College; Visiting Obstetrician, Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 1621 Spruce Street.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital. 1121 Medical Arts Building.

‡1894. Bochroch, Max H., M.D., Professor of Nervous and Mental Diseases, Temple University; Visiting Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1929. Bockus, Harry L., B.S., M.D., Professor of Gastroenterology, Graduate School of Medicine, University of Pennsylvania, and Attending Gastroenterologist, Graduate Hospital; Attending Physician, Presbyterian Hospital. 250 S. Eighteenth St.

ELECTED

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital. 2213 N. Broad St.

1928. Boles, Russell S., M.D., Associate in Medicine, University of Pennsylvania; Visiting Physician, Philadelphia General Hospital; Consulting Gastroenterologist, U. S. Veterans' Bureau, Philadelphia. Rittenhouse Plaza.

1923. Bond, Earl D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, School of Medicine and Graduate School of Medicine, University of Pennsylvania. Bryn Mawr, Pa.

1911. Bonney, Charles W., A.B., M.D., Assistant Professor of Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1924. Boon, David J., M.D., Opthalmologist, Memorial Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Ophthalmic Surgeon, Episcopal Hospital. 1532 N. Fifteenth Street.

1931. Bortz, Edward L., A.B., M.D., Visiting Physician to Lankenau Hospital; Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Chief of Medical Service "B," Lankenau Hospital. 2021 W. Girard Ave.

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Department, Jefferson Medical College; X-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 4940 Penn St.

1928. Bothe, Albert Edward, B.S., M.D., M.S., Instructor in Surgery, Medical School and Instructor in Urology, Graduate School, University of Pennsylvania; Assistant in Urology, Presbyterian Hospital; Urologist, Misericordia Hospital. 133 S. Thirty-sixth St.

1928. Bothe, Frederick A., M.S., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Presbyterian Hospital; Associate Surgeon, Children's Hospital; Surgeon to Stetson Hospital. 133 S. Thirty-sixth St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital. 2509 Boardwalk, Atlantic City, N. J.

1923. Bower, John O., M.D., Surgeon to the Philadelphia General and Northeastern Hospitals; Consulting Surgeon to United States Veterans' Hospital No. 49; Clinical Professor of Surgical Research, Temple University. 2008 Walnut St.

ELECTED

1891. Boyd, George, M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Consulting Obstetrician to the Preston Retreat and Philadelphia General Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Medical Electrician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1925. Boykin, Irvine M., M.D., Associate Surgeon to the Episcopal Hospital; Associate in Surgery, Graduate School of Medicine, Instructor in Surgery, School of Medicine, University of Pennsylvania. 922 Medical Arts Building.

1929. Bradbury, Samuel, M.D., Medical Director, Out-Patient Department, Pennsylvania Hospital; Chief of Service, "B," Department of Medicine, Germantown Dispensary and Hospital. 151 W. Coulter St.

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Associate Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

1921. Bransfield, John W., M.D., Clinical Professor of Surgery, Woman's Medical College; Visiting Surgeon to St. Agnes, St. Vincent's and Woman's College Hospitals. 2101 Spruce Street.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1900. Brinton, Ward, A.M., M.D., Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Visiting Physician, Department for Tuberculosis, Philadelphia General Hospital, and Eaglesville Sanatorium. 1423 Spruce St.

1917. Bromer, Ralph S., A.B., M.D., Roentgenologist to Bryn Mawr Hospital; Associate in Radiology, Graduate School of Medicine, University of Pennsylvania. Bryn Mawr, Pa.

1932. Brown, Claude P., M.D., Pathologist, Veterans' Bureau Hospital. 1930 Chestnut St.

1919. Brown, Henry P. Jr., M.D., Surgeon and Chief of Out-Patient Clinic of the Pennsylvania Hospital; Surgeon to the Children's Hospital; Associate Surgeon, Presbyterian Hospital; Assistant Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1930 Chestnut St.

ELECTED

1916. Brown, Samuel Horton, M.D. 1930 Chestnut St.

1887. Brubaker, Albert P., A.M., M.D., Emeritus Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1930. Bucher, Carl J., B.S., M.D. The Westbury, Fifteenth and Spruce Sts.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Professor of Psychiatry, Graduate School of Medicine, and Associate Professor of Psychiatry, School of Medicine, University of Pennsylvania; Neurologist to Frankford Hospital; Honorary Consultant to the Psychopathic Department, Philadelphia General Hospital. Frankford, Phila.

1928. Burden, Verne G., M.D., M.S., Surgeon to St. Joseph's and Philadelphia General Hospitals; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. The Warwick, Seventeenth and Locust Sts.

1927. Burns, Michael A., M.D., Associate Professor Nervous and Mental Diseases, Jefferson Medical College; Chief of the Nervous and Mental Clinic, Jefferson Hospital; Visiting Neurologist, Philadelphia General Hospital; Neuropsychiatrist, St. Mary's Hospital. N. E. Cor. Twentieth and Chestnut Sts.

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1527 Pine St.

1906. Butler, Ralph, M.D., Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 1930 Chestnut St.

*1908. Cadbury, William W., A.M., M.D., Internist, Canton Hospital, Lingnan University. Canton, China.

†1907. Cadwalader, Williams B., M.D., Clinical Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 133 S. Thirty-sixth St.

1928. Campbell, Edward H., M.D., Assistant Oto-laryngologist, Pennsylvania and Children's Hospitals, and in Out-Patient Department of Lankenau Hospital; Instructor in Oto-laryngology in the Medical School and Graduate School of Medicine, University of Pennsylvania. 2117 Chestnut St.

ELECTED

1931. Cantarow, Abraham, M.D., Instructor in Medicine, Jefferson Medical College; in charge of Laboratory of Biochemistry, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital. 2031 Pine St.

1931. Cariss, Walter L., A.B., M.D., Associate in Oto-laryngology, Presbyterian Hospital. 2043 Walnut St.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

1930. Carpenter, Chapin, A.B., M.D., Assistant Ophthalmologist, Bryn Mawr Hospital; Surgeon, Eye Dispensary, University Hospital; Instructor in Ophthalmology, Under-Graduate Department, University of Pennsylvania. 1930 Chestnut St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1926. Carpenter, Howard Childs, M.D., Vice-Dean for Pediatrics and Professor of Pediatric Hygiene, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania; Director, Department for the Prevention of Disease, Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Consulting Ophthalmologist to the Bryn Mawr and Abington Hospitals. 1930 Chestnut St.

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine St.

1922. Case, Eugene A., M.D., Professor of Morbid Anatomy, Graduate School of Medicine, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1892. Cattell, Henry W., A.M., M.D., Editor of the International Clinics. Box 311, Burlington, N. J.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 315 S. Fifteenth St.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

ELECTED

1929. Clark, Eliot R., A.B., M.D., Professor of Anatomy and Director of the Department of Anatomy, School of Medicine, University of Pennsylvania. 315 S. Forty-first St.

1926. Clarke, J. Alexander, A.B., M.D., Chief Clinical Assistant, Out-Patient Department of Applied Immunology, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital; Instructor in Medicine, Undergraduate School, University of Pennsylvania. 334 S. Twenty-first St.

1926. Clerf, Louis H., M.D., Professor of Bronchoscopy and Esophagoscopy, Jefferson Medical College; Attending Bronchoscopist, Jefferson Hospital; Bronchoscopist, Pennsylvania, Germantown, Jewish and St. Joseph's Hospitals. 126 S. Tenth St.

1910. Cloud, J. Howard, M.D., Attending Physician to the Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Oto-Rhinology, Graduate School of Medicine, University of Pennsylvania; Surgeon in Charge Department Oto-laryngology, Presbyterian and Abington Memorial Hospitals; Associate Laryngologist, Pennsylvania Hospital; Consulting Oto-laryngologist to the Philadelphia Orphanage, U. S. Veterans' Bureau and Base Hospital No. 49, Chester County Hospital, Lawrenceville School and to the Sharon Hospital, Sharon, Conn. 1721 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. S. E. Cor. Forty-second and Spruce Sts.

1922. Cohen, Leon Solis, A.B., M.D., Chief Clinical Assistant of Roentgenology, Jefferson Medical College; Radiologist to the Memorial Hospital, Roxborough; Radiologist and Director of the Research Tumor Clinic, Jewish Hospital. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence and Toxicology, Woman's Medical College of Pennsylvania; Pediatrist to the Jewish and Mount Sinai Hospitals. 2110 Spruce St.

1888. Cohen, Solomon Solis, M.D., H.L.D., Emeritus Professor of Clinical Medicine in the Jefferson Medical College; Emeritus Physician to the Jefferson Hospital; Consulting Physician to the Philadelphia General and Jewish Hospitals. 1906 Walnut Street.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Physician to Out-Patient Department, Mary Drexel Home. Elkins Park, Pa.

ELECTED

1898. Coles, Stricker, M.D. Bryn Mawr, Pa.

1930. Cooper, David A., B.S., M.D., Instructor in Medicine, University of Pennsylvania. 1520 Spruce St.

‡1932. Cope, Thomas A., M.D.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. Twenty-first and Vine Sts.

1914. Corson, Edward Foulke, M.D., Assistant Professor of Dermatology, Jefferson Medical College; Chief Clinical Assistant in Dermatology, Jefferson Hospital; Dermatologist, Chestnut Hill Hospital. Medical Arts Building.

1930. Cottrell, James E., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant Physician, University Hospital; Associate Physician, Episcopal Hospital. 256 S. Twenty-first St.

1924. Cowan, Alfred, M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. Henry Phipps Institute.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Philadelphia Orphanage. 2031 Locust St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service, Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

ELECTED

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. Box 126, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania; Assistant Surgeon Orthopedic Hospital. 5324 Wayne Ave.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 406 S. Twenty-second St.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2047 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-Patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth St.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1916. Davis, Warren B., M.D., Assistant Professor of Oral Surgery, Jefferson Medical College; Chief of Maxillo-facial Surgery Clinic, Jefferson Hospital; Consulting Oto-laryngologist, St. Agnes' Hospital; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D., Physician to the Rush Hospital; Clinical Chief, City Chest Clinic, No. 12. Rush Hospital.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

ELECTED

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor in Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1923. De Long, Perce, M.D., Associate in Ophthalmic Pathology in the Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Wills Eye Hospital; Assistant Neuropathologist, Philadelphia General Hospital. 1904 Chestnut Street.

1928. Dewees, A. Lovett, B.A., M.D., Attending Physician, Bryn Mawr Hospital. Times Medical Building, Ardmore, Pa.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 1600 Professional Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-Patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth Street.

1924. Dillon, Edward S., A.B., M.D., Chief of Division of Metabolism, Philadelphia General Hospital; Assistant Physician, Pennsylvania Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania. 1803 Pine St.

1932. Dintenfass, Henry, M.D. 1305 Spruce St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 326 S. Nineteenth St.

1920. Doane, Joseph Chapman, M.D., Associate Professor of Medicine, Temple University; Medical Director, Jewish Hospital; Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. Nineteenth and Spruce Sts.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital. 185 N. Wabash Ave., Chicago, Ill.

1907. Dorrance, George Morris, M.D., Professor of Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Surgeon to St. Agnes' Hospital. 2101 Spruce St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

ELECTED

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Pennsylvania Institute for the Instruction of the Blind. 407 S. Twenty-second Street.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1924. Egbert, Seneca, M.D., Professor of Hygiene, School of Hygiene and Public Health, University of Pennsylvania. Wayne, Pa.

1930. Eiman, John, M.D., Director, Department of Pathology, Presbyterian Hospital; Assistant Professor of Pathology, Graduate School and Associate Department of Pathology, School of Medicine, University of Pennsylvania. Cynwyd, Pa.

1911. Eliason, Eldridge L., B.A., M.D., Sc.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital and to the Howard Hospital; Assistant Surgeon to the Philadelphia General Hospital and the American Stomach Hospital. 326 S. Nineteenth St.

1928. Ellison, Richard T., B.S., M.D., Instructor in Medicine, School of Medicine, University of Pennsylvania; Assistant Physician in Charge of Heliotherapy at the Home for Consumptives, Chestnut Hill; Assistant Physician, Presbyterian Hospital. 111 Rex Ave.

1904. Elmer, Walter G., B.S., M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Hospital of the Graduate School of Medicine and to the Philadelphia General Hospital. 1801 Pine St.

1932. Engel, Gilson Colby, A.B., M.D., Assistant Surgeon, Germantown, Lankenau and Mary J. Drexel Hospitals; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. 1914 Pine St.

†1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

ELECTED

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Medicine, Medical School, University of Wisconsin; Physician to State of Wisconsin General Hospital; Consulting Physician, Madison General and St. Mary's Hospitals. 1300 University Ave., Madison, Wis.

1905. Evans, William, M.D. 500 W. Gay St., West Chester.

1912. Eves, Curtis C., M.D., Chief of Oto-laryngological Service, Pennsylvania Hospital; Oto-laryngologist to Bryn Mawr Hospital; Consultant Oto-laryngologist to the Philadelphia General Hospital. 1910 Spruce St.

1922. Farley, David L., B.S., M.D., Associate in Medicine, University of Pennsylvania; Physician to Pennsylvania Hospital; Chief of Out-Patient Medical Clinic, Pennsylvania Hospital. 1725 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 121 Maplewood Ave., Germantown.

1930. Farrell, John T., Jr., B.S., M.D., Assistant Roentgenologist, Jefferson Hospital; Roentgenologist to Daniel Baugh Institute of Anatomy of Jefferson Medical College. 235 S. Fifteenth St.

‡1907. Fetterolf, George, A.B., M.D., Sc.D., Professor of Oto-laryngology, School of Medicine, University of Pennsylvania. 2010 Spruce St.

1924. Fewell, Alexander G., M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, and Instructor in Ophthalmology in the School of Medicine, University of Pennsylvania; Ophthalmic Surgeon to the Episcopal Hospital. 1924 Pine St.

1907. Fife, Charles A., A.B., M.D., Associate Professor of Pediatrics in the Graduate School of Medicine of the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., A.B., M.D. Jenkintown, Pa.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes' and Pottstown Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

ELECTED

1928. Fitz-Hugh, Thomas, Jr., A.B., A.M., M.D., Associate in Medicine, Medical School, University of Pennsylvania; Chief of Hematology Section, Medical Clinic, University Hospital. 2020 Locust St.

1928. Flick, John Bernard, M.D., Surgeon to Pennsylvania Hospital; Assistant Surgeon to Bryn Mawr Hospital; Associate in Surgery, Jefferson Medical College. 1608 Spruce St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Clinical Laboratory and Professor of Comparative Pathology, School of Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Director of Pathological Service, Germantown Hospital. Haverford, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania. Overbrook, Pa.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 3400 Spruce St.

1928. Fuller, Daniel Hunt, A.B., A.M., M.D., Medical Director, Department for Men, Pennsylvania Hospital for Mental and Nervous Diseases. 4401 Market St.

‡1916. Funk, Elmer Hendricks, M.D., Clinical Professor of Medicine and Therapeutics in the Jefferson Medical College; Assistant Physician to the Pennsylvania and Jefferson Hospitals; Visiting Physician to the White Haven Sanatorium. 269 S. Nineteenth St.

1899. Gamble, Robert, G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1931. Garner, Vaughn C., M.D., M.S., Assistant Professor of Dermatology and Syphilology, University of Pennsylvania. Germantown Professional Building.

*1913. Gates, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

1917. Gerhard, Arthur Howell, M.D. 1615 N. Delaware Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899. Gibbon, John H., M.D., Emeritus Professor of Surgery in the Jefferson Medical College; Consulting Surgeon to the Pennsylvania and Bryn Mawr Hospitals. 1608 Spruce St.

ELECTED

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopedics, University of Pennsylvania; Surgeon to the Orthopedic Hospital; Orthopedic Surgeon to the Presbyterian Hospital; Chief Surgeon to the Widener Memorial Industrial Training School for Crippled Children. 1930 Chestnut St.

1931. Gilman, Robert Louis, B.S., M.Sc. (Med.), M.D., Research Assistant, Department of Dermatological Research, University of Pennsylvania; Assistant Professor of Dermatology, Woman's Medical College. 1930 Chestnut St.

1927. Gilmour, William Rodger, A.B., A.M., M.D., Assistant Surgeon, Philadelphia General and Methodist Hospitals; Instructor in Anatomy, University of Pennsylvania; Associate Professor of Operative Surgery and Surgical Anatomy, Woman's Medical College. 1737 Chestnut St.

1931. Giordano, Anthony A. S., M.D., Instructor in Oto-laryngology, School of Medicine, University of Pennsylvania; Instructor in Otology, Graduate School of Medicine, University of Pennsylvania; Assistant in Oto-laryngology, St. Agnes Hospital. 1819 S. Broad St.

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D., Chief Pharmacologist, H. K. Mulford Company. The Cambridge, Wissahickon and School Lane, Germantown.

*1906. Gittings, J. Claxton, M.D. Harwood P.O., Md.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania. 2112 Pine St.

1906. Goepp, R. Max, M.D. 518 Medical Arts Building.

1906. von Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal Hospital and Baltimore & Ohio Railroad; Consulting Ophthalmologist, Misericordia and Kensington Hospitals. 2213 Walnut St.

*1908. Goodman, Edward H., M.D. Dorset, Vt.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

1928. Gordon, Burgess Lee, A.B., M.D., Associate Professor in Medicine, Jefferson Medical College; Director of the Depart-

ELECTED

ment for Diseases of the Chest, Jefferson Hospital; Visiting Physician to White Haven Sanatorium and Out-patient Department, Pennsylvania Hospital. 1832 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1926. Grant, Francis C., A.B., M.D., Assistant Professor of Neurosurgery and Associate in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital. 3400 Spruce St.

1904. Grayson, Charles P., M.D., Emeritus Professor of Laryngology in the University of Pennsylvania; Oto-laryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1932. Griffith, George Cupp, A.B., M.D., Associate Physician and Director, Department of Diseases of the Heart, Presbyterian Hospital; Assistant Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania. 4005 Chestnut St.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the Graduate School of Medicine, University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital. 2213 Walnut St.

1932. Guequierre, Jacques P., M.D., B.S., M.Sc., Associate in Dermatology, Graduate School of Medicine, University of Pennsylvania; Lecturer in Dermatology, Temple University School of Medicine; Dermatologist to Philadelphia Hospital for Mental Diseases. 1930 Chestnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty, University of Toronto School. 109 Madison Ave., Toronto, Canada.

ELECTED

1894. Hamill, Samuel McC., M.D., Consulting Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Children's Hospital; Visiting Physician to the Children's Hospital of the Mary J. Drexel Home; Pediatrist to the Widener Memorial School; Consulting Pediatrist to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College. N. E. Cor. Seventeenth and Walnut Sts.

1932. Harris, Stanley E., A.B., M.D., Physician, Student Health Service, University of Pennsylvania; Assistant Medical Service "B" and Cardiovascular Section, Episcopal Hospital; Staff, Delaware County Hospital, Upper Darby. Lansdowne, Pa.

1929. Harrison, Francis G., M.D., Assistant Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Assistant Urologist, Out-Patient Department, Presbyterian Hospital. 1900 Spruce St.

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

1926. Hartman, Frederick L., A.B., M.D., Chief, Medical Service "A," Lankenau Hospital. 1914 Pine St.

1932. Hartman, John Calvin, M.D. 100 W. Walnut Lane.

1907. Hatfield, Charles James, A.B., M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

*1928. Hayman, Joseph M., Jr., A.B., M.D., Associate Professor of Medicine, Western Reserve University; Associate Physician, Lakeside Hospital. Cleveland, Ohio.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's

ELECTED

Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital; Director of Public Health, Philadelphia. 1906 Spruce St.

1927. Herman, Leon, B.S., M.D., Urologist, Methodist Hospital; Assistant Surgeon in Charge of Urology, Pennsylvania Hospital. 801 Medical Arts Building.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 257 S. Sixteenth St.

1924. Hewson, William, A.B., M.D., Instructor in Otology, Graduate School of Medicine, University of Pennsylvania; Oto-laryngologist, Pennsylvania Institution for Instruction of Blind; Out-Patient Department, Children's Hospital; Visiting Surgeon Department Oto-laryngology, Abington Memorial Hospital. 6013 Greene St., Germantown.

*1892. Hinsdale, Guy, A.M., M.D., Medical Director, White Sulphur Springs, W. Va.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Gynecologist to the Philadelphia General Hospital. 1821 Spruce St.

1924. Hirst, John C., 2nd, A.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Assistant Obstetrician, University Hospital; Obstetrician to St. Agnes' Hospital; Gynecologist to Orthopedic Hospital. 1918 Pine St.

*1908. Hitchens, Arthur Parker, M.D., M.C., U.S.A., in charge, 6th Corps Area. Laboratory, Fort Sheridan, Ill.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian Hospital; Chief of Surgical Service, Germantown Hospital; Surgeon-in-Chief to Chester County Hospital, West Chester, Pa. 2019 Spruce St.

1931. Holland, H. Albert, M.D., Assistant Chief, Medical Staff, Chestnut Hill Hospital. 8200 Crittenden St., Chestnut Hill.

1932. Hollingsworth, I. Pemberton P., M.D., Pathologist to Chester County Hospital. West Chester, Pa.

1906. Holloway, Thomas B., M.S., M.D., Sc.D., Professor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Orthopedic Hospital, and in Department of Neurology, Philadelphia General Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut St.

1919. Hopkins, Arthur H., B.S., M.D. 1726 Pine St.

ELECTED

1925. Hopkinson, R. Dale, M.D., Registrar and Assistant Physician, Abington Memorial Hospital. Jenkintown, Pa.

1928. Houser, Karl M., Ph.B., M.D., M.Sc., Instructor in Oto-laryngology, University of Pennsylvania; Assistant in Oto-laryngology, University Hospital; Oto-laryngologist, Delaware County Hospital. 2010 Spruce St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories and Dean of the Graduate School, University of Michigan. 1330 Hill St., Ann Arbor, Mich.

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D. N. E. Cor. Twentieth and Chestnut Sts.

*1912. Hunter, John W., B.S., M.D. McDaniel, Md.

1921. Hunter, Robert J., M.D., Laryngologist, Philadelphia General Hospital; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. 2011 Chestnut Street.

1926. Husik, David N., M.D., Assistant Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Attending Laryngologist, Philadelphia General and Mount Sinai Hospitals; Consulting Oto-laryngologist, American Stomach Hospital. 1930 Chestnut St.

1898. Hutchinson, James P., M.D., Consulting Surgeon to the Pennsylvania and to the Methodist Hospitals. Media, Pa.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Consulting Plastic Surgeon to Children's and Chestnut Hill Hospitals; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D. C. 1930 Chestnut St.

1917. Jackson, Chevalier, M.D., Sc.D., LL.D., Professor of Bronchoscopy and Esophagoscopy, Temple University School of Medicine; Bronchoscopist and Esophagoscopist, Temple University Hospital; William Potter Memorial Lecturer, Jefferson Medical College; Lecturer on Bronchoscopy and Esophagoscopy, Woman's Medical College. 3432 N. Broad St.

ELECTED

1932. Jackson, Chevalier Lawrence, A.B., M.D., M.Sc., Clinical Professor of Bronchoscopy and Esophagoscopy, Temple University School of Medicine; Associate in Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania; Bronchoscopist, Chestnut Hill Hospital. 3432 N. Broad St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Secretary of State Commission for the Blind of Colorado. 217 Imperial Building, Denver, Colo.

‡1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Chester County Hospital. West Chester, Pa.

1922. Jameson, Howard Leon, M.D., Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2133 S. Sixteenth St.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. Rosemont, Pa.

1930. Jepson, Paul Newton, A.B., M.D., M.S. 1824 Spruce St.

1928. John, Rutherford Lewis, B.S., M.D., Instructor in Surgery, School of Medicine, University of Pennsylvania; Associate Surgeon in Charge of Orthopedics, Episcopal Hospital; Orthopedic Surgeon to St. Christopher's Hospital; Assistant Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases. 256 S. Twenty-first St.

1926. Johnson, George Ernest, M.D. 5341 Chester Ave.

1898. Johnson, Russell H., A.B., M.D., Physician to the Pennsylvania School for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1918. Jonas, Leon, M.D. 1726 Pine St.

1924. Jones, Harold W., M.D., Demonstrator of Medicine, Jefferson Medical College; Assistant Physician, Philadelphia General Hospital. 1930 Chestnut St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 2056 N. Sixty-third St.

1900. Jopson, John H., M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to Children's and Bryn Mawr Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-Patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

ELECTED

1902. JUMP, HENRY D., M.D., Professor of Applied Therapeutics and Clinical Medicine, Woman's Medical College; Physician to Philadelphia General and Misericordia Hospitals. 2019 Walnut St.

1903. KALTEYER, FREDERICK J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. KARSNER, HOWARD T., M.D., Professor of Pathology and Director of the Institute of Pathology, Western Reserve University. 2109 Adelbert Rd., Cleveland, Ohio.

1926. KAUFMAN, ISADORE, M.D., Associate in Medicine, University of Pennsylvania; Medical Director, Kensington Dispensary for Tuberculosis; Associate Clinical and Sociological Director, Phipps Institute; Visiting Physician to White Haven Sanatorium. 1616 Walnut St.

1923. KAY, JAMES, M.D., Visiting Physician, Episcopal Hospital and to the Philadelphia Hospital for Mental Diseases, Byberry; Associate Professor in Medicine, Medical School, Temple University; Consulting Physician to Kensington Hospital for Women. 600 W. Olney Ave.

1924. KEELER, J. CLARENCE, M.D., Associate Professor of Otology, Jefferson Medical College and Hospital; Assistant in Otolaryngology, Germantown Hospital; Consultant Otolaryngologist, Pottstown Hospital, Pa., and Newcombe Hospital, Vineland, N. J. Medical Arts Building.

‡1867. KEEN, WILLIAM W., M.D., LL.D. (Hon.), Ph.D. (Brown, Upsala, etc.), (Hon.) Sc.D. (Jefferson and Harvard), Dr., Honoris Causa, University of Paris; (Hon.) F.R.C.S. (Eng., Edin. and Ireland). 1520 Spruce St.

1912. KEENE, FLOYD E., M.D., Goodell Professor of Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. 133 S. Thirty-sixth St.

1913. KELLY, FRANCIS JOSEPH, M.D. 2211 Locust St.

1932. KELLY, HERBERT T., M.D., Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. 1900 Spruce St.

*1887. KELLY, HOWARD A., A.B., M.D., LL.D., Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the

ELECTED

Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. KELLY, JAMES A., A.M., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon to St. Mary's, St. Joseph's and Misericordia Hospitals. 1815 Spruce St.

1912. KELLY, THOMAS C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. KEMPTON, AUGUSTUS F., M.D. 2118 Pine St.

1905. KERCHER, DELNO E., M.D. 1534 Pine St.

1921. KERN, RICHARD A., A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

1930. KIMBROUGH, ROBERT A., JR., A.B., M.D., Associate in Obstetrics and Gynecology, University of Pennsylvania; Assistant Gynecologist, University Hospital; Obstetrician, Pennsylvania Hospital. 1907 Spruce St.

1931. KINNEY, WILLARD H., M.D., Genito-urinary Surgeon, Philadelphia General Hospital; Assistant Attending Genito-urinary Surgeon, Jefferson Hospital; Associate Professor Genito-urinary Surgery, Jefferson Medical College. 315 S. Seventeenth St.

1920. KLAUDER, JOSEPH VICTOR, A.B., M.D., Professor of Dermatology, Woman's Medical College; Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Dermatologist, Misericordia, Wills and Abington Memorial Hospitals; Chief of Clinic, Philadelphia General Hospital. 1934 Spruce St.

1920. KLEIN, THOMAS, M.D., Professor of Applied Therapeutics, Medical School, Temple University; Associate Professor of Medicine, Graduate School, University of Pennsylvania; Physician to Samaritan and Philadelphia General Hospitals. 250 S. Eighteenth St.

1913. KLOPP, EDWARD J., M.D., Professor of Surgery in the Jefferson Medical College; Attending Surgeon to the Jefferson Hospital; Surgeon and Chief of the Out-Patient Clinic of the Pennsyl-

ELECTED

vania Hospital; Surgeon to the Delaware County and Memorial Hospitals. 1611 Spruce St.

1932. Klopp, John W., M.D. 618 W. Lehigh Ave.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish and Mary J. Drexel Hospitals; Associate in Ophthalmology, Jefferson Medical College; Chief of Eye Clinic, Jefferson Hospital. 1737 Chestnut St.

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 1930 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2035 Spruce St.

1925. Knox, Harry E., M.D., Surgeon to St. Christopher's Hospital; Surgeon to Philadelphia Hospital for Contagious Diseases; Associate Surgeon, Episcopal Hospital; Assistant Surgeon, Germantown Hospital. 719 Sixty-sixth Ave., Oak Lane, Phila.

1914. Kolmer, John A., M.D., Dr. P. H., M.Sc., D.Sc., LL.D., Professor of Medicine, Temple University; Head of the Department of Pathology and Bacteriology, Research Institute for Cutaneous Medicine. Cynwyd, Pa.

1932. Kornblum, Karl, B.S., M.D. Wynnewood, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's Hospital for Children; Laryngologist to the Abington Hospital. 1930 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Professor of Pathology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. Lansdowne, Pa.

1932. Landis, Eugene M., A.B., M.S., M.D., Ph.D., Associate in Medicine, University of Pennsylvania, University Hospital. Thirty-sixth and Spruce Sts.

1904. Landis, Henry R. M., M.D., Professor of Clinical Medicine in the School of Medicine, and Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

ELECTED

1907. Langdon, H. Maxwell, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist of the Presbyterian, Orthopedic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 405 S. Forty-second St.

1926. Laws, George M., B.S., M.D., Associate Gynecologist, Presbyterian Hospital; Gynecologist, American Hospital for Diseases of the Stomach. 1907 Spruce St.

1920. Leavitt, Frederick Headley, M.D., Associate in Neurology, Graduate School of Medicine, University of Pennsylvania; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Assistant Professor, Epidemic Contagious Diseases, Graduate School of Medicine, University of Pennsylvania; Senior Pediatrist, Philadelphia Hospital for Contagious Diseases. 1930 Chestnut St.

1908. Lee, Walter Estell, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr, Children's and Burlington County Hospitals, and to the Hospital of the Graduate School of Medicine, University of Pennsylvania. 905 Pine St.

‡1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1713 Walnut St.

1920. Leopold, Simon S., M.D., Assistant Professor of Medicine and Head of the Department of Physical Diagnosis, University of Pennsylvania; Chief of Respiratory Section and Assistant Physician, University Hospital. 2025 Spruce St.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 259 S. Seventeenth St.

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Neurology, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

1927. Lindsey, Walter H., A.B., M.D., Associate Physician, Episcopal Hospital; Assistant Medical Director, Philadelphia

ELECTED

Rapid Transit Company. S. E. Cor. Seventeenth and Arch Streets.

‡1886. Llyod, J. Hendrie, A.M., M.D., Consulting Neurologist to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1931. Loewenberg, Samuel A., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Visiting Physician Jefferson Hospital; Visiting Physician, Philadelphia General and Northern Liberties Hospitals and to Eaglesville Sanitorium. 1905 Spruce St.

1893. Longaker, Daniel, M.D., Consulting Obstetrician to the Kensington Hospital for Women. 1806 Spruce St.

1931. Longaker, Edwin P., M.D., Instructor in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Laryngologist, Bryn Mawr Hospital; Assistant Larynologist and Otologist, Out-Patient Department, Pennsylvania Hospital. 1806 Spruce St.

1921. Lucké, Baldwin, Dr. P.H., M.D., Professor of Pathology, School of Medicine, University of Pennsylvania; Pathologist, Philadelphia General Hospital. Medical School, University of Pennsylvania.

1924. Ludy, John B., A.B., A.M., M.D., Dermatologist to Pennsylvania, Lankenau, Episcopal, Methodist, Philadelphia General, Delaware County and Oncologic Hospitals. Medical Arts Building.

1923. Lueders, Charles W., A.B., M.D., Gastroenterologist and Chief of Clinic, Pennsylvania Hospital. 1930 Chestnut St.

1932. Lukens, Francis D. W., A.B., M.D., Instructor in Medicine, University of Pennsylvania and Associate in the George S. Cox Medical Research Institute, University of Pennsylvania, University Hospital. Thirty-sixth and Spruce Sts.

1930. Lukens, Robert M., M.D., Associate in Bronchoscopy and Esophagoscopy, Jefferson Medical College; Assistant Bronchoscopist, Jefferson and Jewish Hospitals; Laryngologist, Henry Phipps Institute. 1923 Spruce St.

1929. Lull, Clifford B., M.D., Associate in Obstetrics, Jefferson Medical College; Attending Obstetrician and Gynecologist, Philadelphia General Hospital; Assistant Obstetrician, Lying-

ELECTED

in Hospital; Director in Obstetrics and Gynecology, Delaware County Hospital. 1731 Pine St.

1919. LYNCH, FRANK B., JR., M.D., Chief of Pathological Division, Germantown Dispensary and Hospital; Associate in Medicine, University of Pennsylvania, Germantown Hospital. E. Wister St.

1914. LYON, B. B. VINCENT, A.B., and D.Sc., M.D., Assistant Professor of Medicine, Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department of the Curtis Clinic, Jefferson Hospital. 2031 Locust St.

1932. MCCAHEY, JAMES FRANCIS, M.D., Assistant Demonstrator of Urology, Jefferson Medical College; Assistant Urologic Surgeon, Philadelphia General Hospital; Urologist, Misericordia Hospital. 1709 Pine St.

1900. MCCARTHY, DANIEL J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1931. MCCLOSKEY, EDWARD, M.D. 7 E. Chestnut Ave.

1913. MCCRAE, THOMAS, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce St.

1895. MCFARLAND, JOSEPH, M.D., Sc.D., Professor of Pathology in the School of Medicine, University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 542 W. Hortter St., Germantown.

1931. MCIVER, JOSEPH, M.D., Associate Neurologist, University of Pennsylvania; Neurologist, Misericordia Hospital; Visiting Psychiatrist, Philadelphia General Hospital. 133 S. Thirty-sixth St.

1905. MCKENZIE, R. TAIT, M.D., LL.D., Emeritus Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine St.

*1916. MCKNIGHT, HOWARD A., A.B., M.D. Tecumseh Building, Springfield, Ohio.

1924. MCMILLAN, THOMAS M., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Cardiologist, Philadelphia General Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania; Associate Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania. 2044 Locust St.

ELECTED

1924. McPhedran, F. Maurice, A.B., M.D., Associate in Medicine, School of Medicine, University of Pennsylvania; Assistant Professor of Medicine, Henry Phipps Institute. Seventh and Lombard Sts.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

1932. Macfarlane, Catharine, M.D., Professor of Gynecology, Woman's Medical College; Gynecologist in Chief, Hospital of Woman's Medical College; Gynecologist, Woman's Hospital; Gynecologist and Obstetrician, Philadelphia General Hospital. Medical Arts Building.

1910. Mackinney, William H., M.D., Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Philadelphia General Hospital; Chief of Urologic Clinic, Lankenau Hospital. 1930 Chestnut St.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Manges, Willis F., D.Sc., M.D., Roentgenologist to the Jefferson Hospital; Clinical Professor of Roentgenology, Jefferson Medical College. 235 S. Fifteenth St.

1898. Marshall George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital; Associate Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania. 1819 Spruce St.

1889. Martin, Edward M.D., Emeritus Professor of Surgical Physiology, University of Pennsylvania. 135 S. Eighteenth St.

1932 Meade, Richard H., Jr., B.S., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania. 200 W. Mermaid Lane.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1736 M. St., N. W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Surgeon, Northwestern General Hospital; Associate Surgeon to the Lankenau Hospital. 1816 Spruce St.

1923. Mendelson, Walter, M.D. 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Children's Hospital,

ELECTED

Jewish Hospital, Misericordia Hospital; St. Edmond's Home for Crippled Children; Children's Seashore House, Lock Haven Hospital, City Hospital, Williamsport, Pa., and Geisinger Memorial Hospital, Danville, Pa. 2017 Spruce St.

*1894. MILLER, D. J. MILTON, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. MILLER, EDWIN B., M.D., Ophthalmic Surgeon, Stetson Hospital. 269 S. Nineteenth St.

1932. MILLER, M. VALENTINE, B.S., M.D., Associate in Otology, Graduate School of Medicine, University of Pennsylvania; Oto-laryngologist, Memorial Hospital, Roxborough. 44 W. Phil-Ellena St.

1921. MILLER, T. GRIER, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Chief, Gastro-intestinal Clinic; Assistant Physician, University Hospital; Consultant, Friends' Hospital and Shriners' Hospitals for Crippled Children. 318 University Hospital.

*1917. MITCHELL, A. GRAEME, M.D., Professor of Pediatrics, College of Medicine, University of Cincinnati; Director of Pediatric and Contagious Services, Cincinnati General Hospital; Medical Director and Chief of Staff of Children's Hospital, Cincinnati. University of Cincinnati.

1904. MITCHELL, CHARLES F., M.D., Chief of Service "A," Pennsylvania, Bryn Mawr and Germantown Hospitals; Consulting Surgeon, Chestnut Hill and St. Christopher's Hospitals. 2003 Pine St.

1921. MOHLER, HENRY K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

1932. MOHLER, ROY W., A.B., M.D., Demonstrator of Gynecology, Jefferson Medical College; Assistant Obstetrician, Lying-in Pennsylvania Hospital; Assistant Obstetrician and Gynecologist, Methodist Hospital. 323 S. Twentieth St.

‡1908. MONTGOMERY, CHARLES M., A.B., M.D. Oteen, N. C.

1932. MONTGOMERY, THADDEUS L., A.B., M.D., Associate in Obstetrics, Jefferson Medical College; Assistant Obstetrician, Jefferson Hospital. 1930 Chestnut St.

1928. MOON, VIRGIL HOLLAND, A.B., M.D., M.Sc., Professor of Pathology, Jefferson Medical College; Chairman, U. S. National Committee, International Association for Geographic Pathology. Jefferson Medical College.

ELECTED

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Bronchoscopist to Philadelphia General Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-Patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

1924. Moorhead, Stirling W., A.B., M.D., Assistant Professor of Urology, School of Medicine, University of Pennsylvania; Associate in Urology, Graduate School of Medicine, University of Pennsylvania; Associate Urologist, University Hospital; Consulting Urologist, Montgomery County Hospital. 1523 Pine St.

1918. Morgan, Arthur C., M.D., Emeritus Professor of Clinical Medicine, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Consulting Physician to the Eastern State Penitentiary and to the Frankford Hospital; Member Pennsylvania State Board of Medical Education and Licensure. 1930 Chestnut St.

1886. Morris, Caspar, M.D. Haverford, Pa.

1893. Morris, Elliston J., M.D., Consulting Physician to the Episcopal Hospital. 128 S. Eighteenth St.

1929. Mudd, Stuart, B.S., M.A., M.D., Associate Professor of Bacteriology, School of Medicine, University of Pennsylvania. Haverford, Pa.

1905. Muller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals and Mary J. Drexel Home. 1930 Spruce St.

1930. Murphy, Douglas P., M.D., Fellow in Research, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania. 4312 Pine St.

*1915. Musser, John H., Jr., B.S., M.D., Professor of Medicine and Head of Department, School of Medicine, Tulane University of Louisiana. 1427 Second St., New Orleans, La.

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Associate Surgeon to the Orthopædic Hospital. 1625 Spruce Street.

1902. Nassau, Charles F., M.D., LL.D., Sc.D., Clinical Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College. 1710 Locust St.

ELECTED

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Consultant Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. Radnor, Pa.

1905. Norris, Charles C., M.D., Director of the Department and Professor of Obstetrics and Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist and Obstetrician to the University Hospital; Director, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania; Gynecologist to Henry Phipps Institute and to the Children's Hospital. 133 S. Thirty-sixth St.

*1905. Norris, George William, A.B., M.D., U. S. Public Health Service (Veterans' Bureau). Dimock, Pa.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Waverly Mills, S. C.

1892. Norris, Richard C., M.D., Professor of Obstetrics in the Graduate School of Medicine, University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1924. Opie, Eugene L., A.B., M.D., Professor of Pathology and Director of the Department of Pathology in the School of

ELECTED

Medicine, University of Pennsylvania; Director of Laboratories, Henry Phipps Institute. Phipps Institute, Seventh and Lombard Sts.

1913. Outerbridge, George W., A.B., M.D., Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Professor of Clinical Surgery, Woman's Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Orthopedic Hospital; Chief Surgeon, Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Consulting Laryngologist, Pennsylvania Hospital; Consulting Oto-laryngologist to the Children's and Bryn Mawr Hospitals, Editor, *Annals of Medical History*. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Chief Medical Service "A," Lankenau Hospital; Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the School of Medicine, University of Pennsylvania and Roentgenologist to the University Hospital. Haverford, Pa.

1909. Parish, Benjamin D., B.S., M.D., Associate Professor in Otology, Graduate School of Medicine, University of Pennsylvania; Aurist and Laryngologist to St. Agnes' and Chestnut Hill Hospitals. 1927 Spruce St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1534 N. Fifteenth St.

1925. Patten, Clarence A., M.D., Associate Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General, Lankenau and Delaware General Hospitals; Consulting Neurologist to Delaware County Hospital. 2037 DeLancey St.

1910. Patterson, Ross Vernet, M.D., Sc.D., Dean, Jefferson Medical College; Honorary Consulting Physician to the Philadelphia General Hospital; Physician-in-charge, Department of Electrocardiology, Jefferson Hospital; Member of Advisory Health Board of Pennsylvania. 2126 Spruce St.

1931. Paul, John Davis, A.B., M.D., Associate Visiting Physician and Associate Physician in charge of Diabetics, Episcopal Hospital; Pathologist to St. Christopher's Hospital. 3112 N. Broad St.

ELECTED

*1926. Paul, John Rodman, A.B., M.D., Assistant Professor of Medicine, School of Medicine, Yale University; Associate Physician, New Haven Hospital and Dispensary. Yale University, New Haven, Conn.

1932. Payne, Franklin L., M.D., Assistant Obstetrician, Pennsylvania Hospital; Assistant Gynecologist, University Hospital; Instructor in Gynecology, University of Pennsylvania. 133 S. Thirty-sixth St.

1922. Pelouze, P. S., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. 1737 Chestnut St.

1909. Pemberton, Ralph, M.S., M.D., Professor of Medicine, Graduate School, University of Pennsylvania; Visiting Physician and Director of the Department of Metabolic Diseases, Presbyterian Hospital; Consultant Physician to Orthopedic Hospital. Paoli, Pa.

1930. Pendergrass, Eugene P., M.D., Assistant Professor of Roentgenology, School of Medicine and Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Assistant Roentgenologist, University Hospital. Ardmore, Pa.

1932. Pennell, Howard Y., M.D., Roentgenologist, Chester County Hospital. West Chester, Pa.

1914. Pepper, O. H. Perry, B.S., M.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

†1902. Pepper, William, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1917. Peter, Luther C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 1930 Chestnut St.

‡1912. Petty, Orlando H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Chief of the Department of Diseases of Metabolism, Hospitals of Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital. 1803 Pine St.

1905. Pfahler, George E., M.D., Sc.D., D.M.R.E. (Camb.), Professor of Radiology and Vice-Dean, Department of Radiology, Graduate School of Medicine, University of Pennsylvania;

ELECTED

Director of Radiological Department, Misericordia Hospital. 1930 Chestnut St.

1915. PFEIFFER, DAMON B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon-in-Chief, Abington Memorial and Lankenau Hospitals. 1822 Pine St.

1907. PFROMM, GEORGE W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 269 S. Nineteenth St.

1907. PHILLIPS, HORACE, M.D., Assistant Surgeon, U. S. Public Health Service; Examining Physician, Office of the District Attorney. 4505 Chester Ave.

1911. PIERSOL, GEORGE MORRIS, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian and Abington Memorial Hospitals; Active Consultant in Medicine, Philadelphia General Hospital. 1913 Spruce St.

1917. PIPER, EDMUND BROWN, B.S., M.D., Professor of Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital; Consulting Obstetrician, Kensington Hospital for Women. 2031 Locust St.

1905. PITFIELD, ROBERT L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. PLEASANTS, HENRY, JR., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopedic Hospital. West Chester, Pa.

1932. PORTER, ROLAND DEL., M.D., Obstetrician-in-Chief, Abington Memorial Hospital; Visiting Surgeon in Gynecology, Abington Memorial Hospital. Jenkintown, Pa.

1896. POSEY, WILLIAM CAMPBELL, M.D., Consulting Ophthalmic Surgeon to Wills, Howard, Chestnut Hill and Abington Hospitals. Radnor, Pa.

*1907. PRICE, GEORGE E., M.D. Stimson Building, Seattle, Wash.

1931. PRYOR, CHARLES ALLEN, M.D., Assistant Oto-laryngologist, Out-Patient Departments of Pennsylvania and Bryn Mawr Hospitals; Assistant in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania. 1910 Spruce St.

*1920. QUICKSALL, WILLIAM E., M.D. 222 Taylor Arcade, St. Petersburg, Fla.

ELECTED

1908. Radcliffe, McCluney, A.M., M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; Consulting Surgeon to the Wills Hospital. 1930 Chestnut St.

1931. Ramsey, Frank M., B.S., M.D., Assistant Attending Physician, Germantown Hospital; Attending Physician, Chestnut Hill Hospital. Chestnut Hill P. O.

1913. Randall, Alexander, A.M., M.D., Associate Professor of Surgery in charge of the Department of Urology, School of Medicine, University of Pennsylvania. 1323 Medical Arts Building.

‡1887. Randall, B. Alex., M.A., M.D., Ph.D., Emeritus Professor of Otology in the School of Medicine, University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb and to Memorial Hospital, Roxborough. 2320 DeLancey St.

1926. Ravdin, I. S., B.S., M.D., J. William White Professor of Surgical Research in the School of Medicine and Assistant Professor of Research Surgery in the Graduate School of Medicine, University of Pennsylvania; Surgeon, University Hospital. 4623 Larchwood Ave.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Westmount Ave., Columbia, Mo.

1928. Reese, Warren S., M.D., Assistant Ophthalmologist, Pennsylvania Hospital; Assistant Surgeon, Wills Hospital; Associate Ophthalmologist, Delaware County Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 230 S. Twenty-first St.

1925. Reeves, Rufus S., B.S., M.D. 2227 Spruce St.

1931. Register, Robert P., M.D., Instructor in Medicine, Jefferson Medical College; Physician to Pennsylvania Hospital; Director Medical Science, Delaware County Hospital. 255 S. Sixteenth St.

1920. Rehfuss, Martin E., M.D. N. E. Cor. Sixteenth and Spruce Streets.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reimann, Stanley P., M.D., Director of the Research Institute, Lankenau Hospital; Associate Professor of Surgical Pathology, Graduate School of Medicine, University of Pennsylvania. 516 Arbutus St., Germantown.

ELECTED

1929. de Rényi, George S., M.D., Associate Professor of Anatomy, University of Pennsylvania. 216 S. Forty-sixth St.

1923. Repplier, Sidney J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

1919. Richardson, Russell, A.M., M.D., Associate in Medicine, University of Pennsylvania and Cox Medical Research Institute, University of Pennsylvania; Director of the Laboratory, Methodist Episcopal Hospital. 320 S. Sixteenth St.

1919. Ridpath, Robert F., M.D., Professor of Rhino-laryngology, Temple University; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Samaritan and Jewish Hospitals. 1737 Chestnut St.

1898. Riesman, David, M.D., Sc.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish, Woman's and Kensington Hospitals. 1520 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Consulting Ophthalmologist to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

1932. Robbins, Frederick R., B.S., M.D., Associate Surgeon, Children's Hospital; Assistant Surgeon, Graduate Hospital of University of Pennsylvania and to Pennsylvania and Bryn Mawr Hospitals. 258 S. Eighteenth St.

1899. Roberts, Walter, M.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1921 Spruce St.

1932. Robertson, Harold F., B.S., M.D., Associate in Medicine, Temple University Hospital; Assistant Visiting Physician, Philadelphia General Hospital; Assistant Syphilologist and Dermatologist, Wills Eye Hospital. 327 S. Seventeenth St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Samaritan and Garretson Hospitals. 327 S. Seventeenth St.

1912. Rodman, John Stewart, M.D., Professor of Surgery, Woman's Medical College; Surgeon-in-Chief, Woman's College Hospital; Attending Surgeon, Bryn Mawr Hospital; Assistant Surgeon to the Presbyterian Hospital. 1310 Medical Arts Building.

ELECTED

1928. Rose, Edward, M.D., Instructor in Medicine, University of Pennsylvania; Medical Registrar, University Hospital. 1726 Pine St.

1909. Rosenberger, Randle C., M.D., Professor of Preventive Medicine and Bacteriology in the Jefferson Medical College. 1009 Walnut St.

1932. Rothrock, Harry A., M.D. West Chester, Pa.

*1907. Royer, B. Franklin, M.D. 450 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., LL.D., James Edwards Professor of Orthopedic Surgery, Jefferson Medical College; Orthopedic Surgeon to the Jefferson Medical College Hospital; Orthopedic Surgeon to the Methodist and the Philadelphia General Hospitals. 911 Medical Arts Building.

1929. Ryan, William John, M.D., Visiting Surgeon to St. Mary's Hospital and to St. Vincent's Hospital for Women and Children. Medical Arts Building.

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Ophthalmic Surgeon to Germantown Hospital. Germantown Professional Building.

1927. Saxon, Gordon J., M.D. 1506 Medical Arts Building.

1906. Saylor, Edwin S., M.D., Consulting Ophthalmologist, American Hospital for Diseases of the Stomach. 1737 Chestnut St.

1920. Scarlett, Hunter W., M.D., Associate in Ophthalmology in the Graduate School of Medicine, and Medical Examiner in the Department of Physical Education, University of Pennsylvania. 230 S. Twenty-first St.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1932. Schaffer, Howard W., M.D. 3475 Midvale Ave.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1929. Scheffey, Lewis C., M.D., P.D., Assistant Gynecologist, Jefferson Hospital; Assistant Professor of Gynecology, Jefferson Medical College. 267 S. Nineteenth St.

1929. Schenck, Harry P., B.S., M.D., Research Fellow in Allergy and Assistant Instructor in Otolaryngology, University of Pennsylvania; Lecturer and Instructor in Pathology, Evans Institute; Assistant Otolaryngologist, Children's Hospital. 1912 Spruce St.

ELECTED

1917. SCHNABEL, TRUMAN GROSS, A.B., M.D., Associate in Medicine, University of Pennsylvania; Physician, Philadelphia General and Medical Dispensary, University Hospital. 1704 Pine St.

1917. SCHOFF, CHARLES H., M.D. Media, Pa.

1928. SCHOFIELD, FREDERICK S., A.B., M.D., Associate in Urology, Medical School, University of Pennsylvania; Chief of Urological Dispensary, University Hospital; Urologist, Orthopedic Hospital; Assistant Urologist, Chestnut Hill Hospital. 822 Medical Arts Building.

1921. SCHUMANN, EDWARD ARMIN, A.B., M.D., Associate Professor of Obstetrics, School of Medicine, University of Pennsylvania; Surgeon-in-Chief, Kensington Hospital for Women, Obstetrician and Gynecologist, Philadelphia General and Memorial Hospitals; Obstetrician, Chestnut Hill Hospital. 1814 Spruce Street.

1887. DE SCHWEINITZ, GEORGE EDMUND, A.M., M.D., LL.D., L.H.D., Sc.D., Honorary Professor of Ophthalmology in the School of Medicine and the Graduate School of Medicine, University of Pennsylvania; Consulting Ophthalmologist to Orthopedic, Philadelphia General and Bryn Mawr Hospitals. 1705 Walnut St.

*1913. DE SCHWEINITZ, GEORGE LORD, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. SCHWENK, PETER N. K., M.A., M.D., Attending Surgeon to Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Hospital. 1417 N. Broad St.

1930. SCOTT, JOHN P., M.D., Associate in Pediatrics, University of Pennsylvania; Pediatrician, Presbyterian Hospital; Assistant Physician, Children's Hospital. 2040 Pine St.

1917. SERVICE, CHARLES A., M.D. Bala, Pa.

1924. SHALLOW, THOMAS A., M.D., Professor of Surgery, Jefferson Medical College. 3942 Chestnut St.

1908. SHANNON, CHARLES E. G., A.B., M.D., Professor of Ophthalmology in the Jefferson Medical College; Attending Ophthalmologist, Jefferson Medical College and Delaware County Hospitals; Ophthalmologist, Presbyterian Orphanage. 1930 Chestnut St.

1928. SHARPE, JOHN SEMPLE, M.D., Attending Physician, Bryn Mawr Hospital. Haverford, Pa.

1928. SHARPLESS, FREDERICK COPE, A.B., M.D., Attending Physician, Bryn Mawr, Hospital. Rosemont, Pa.

ELECTED

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1931. Sheehan, William C., M.D., 8820 Germantown Ave.

1932. Sheridan, Joyce T., B.S., M.D., Assistant Medical Director, The Fidelity Mutual Life Insurance Company. 412 W. Durham St.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. Shoemaker, Harlan, A.B., M.D., Chief Surgeon to Los Angeles County Hospital; Chief of the Surgical Staff of the Methodist Hospital and of the Hospital of the Good Samaritan. 1930 Wilshire Building, Los Angeles, Calif.

†1896. Shoemaker, William T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 2017 Locust St.

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Lankenau and Oncologic Hospitals; Honorary Consultant in Ophthalmology to the Philadelphia General Hospital. 1737 Chestnut St.

1932. Siegel, Alvin E., A.B., M.D., Pediatrician, Methodist Hospital; Physician-in-Chief, Southern Home for Destitute Children; Instructor in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 2021 Spruce St.

1903. Sinclair, John Falconer, M.D., Epidemiologist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1226 Medical Arts Building.

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 2213 DeLancey St.

1928. Skillern, Samuel Ruff, M.D., Associate Professor in Otolaryngology, Graduate School of Medicine, University of Pennsylvania. 1734 Pine St.

1929. Smith, Austin Thomas, M.D., Clinical Assistant, Department of Laryngology, Jefferson Hospital; Instructor in Laryngology, Jefferson Medical College. 259 S. Seventeenth St.

ELECTED

1927. Smyth, Calvin M., Jr., B.S., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon in charge, Methodist Hospital; Visiting Surgeon, Abington Memorial Hospital. 2021 Spruce St.

1919. Smyth, Henry Field, M.D., Assistant Professor of Industrial Hygiene, School of Hygiene, University of Pennsylvania. Wayne, Pa.

1928. Spaeth, Edmund B., M.D., Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, Temple University. 1930 Chestnut St.

1908. Spease, John, M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the Children's Hospital. 133 S. Thirty-sixth St.

1897. Spiller, William G., M.D., Emeritus Professor of Neurology in the University of Pennsylvania; Honorary Consulting Neurologist and Neuropathologist to the Philadelphia General Hospital. 4409 Pine St.

1929. Sponsler, Marshall B., A.B., M.D., Instructor in Oto-laryngology, School of Medicine and Associate in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Oto-laryngologist, Methodist Hospital; Visiting Oto-laryngologist, White Haven Sanatorium. 1934 Spruce St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D. Haverford, Pa.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia, at Wallingford, and to the Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

1927. Starr, Isaac, Jr., B.S., M.D., Assistant Professor of Clinical Pharmacology and Associate in Medicine, School of Medicine, University of Pennsylvania; Acting Physician, University Hospital. Chestnut Hill.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 1900 Rittenhouse Square.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-Patient Surgical Department of the Jefferson Medical College Hospital. 220 S. Sixteenth St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. University Hospital.

ELECTED

1901. Stevens, Arthur A., M.D., Professor of Applied Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to St. Agnes' Hospital. 314 S. Sixteenth St.

1914. Stewart, Thomas S., B.S., M.D. 1532 Pine St.

1931. Stiles, Merritt H., M.D., Assistant in Medicine, in charge Thyroid Dispensary, Germantown Hospital; Assistant in Pediatrics, Chestnut Hill Hospital. 113 W. Chestnut Ave., Chestnut Hill.

1925. Stokes, John H., A.B., M.D., Professor of Dermatology and Syphilology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, University and Philadelphia General Hospitals; Dermatologist, Hospital of the Graduate School of Medicine. 3800 Chestnut St.

1928. Stokes, Joseph, Jr., A.B., M.D., William H. Bennett Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania. 159 W. Coulter St., Germantown.

‡1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society and the William Penn Charter School. 2015 Walnut St.

1923. Strecker, Edward A., A.M., M.D., Professor of Psychiatry, School of Medicine, University of Pennsylvania; Chief of Clinic and Consultant, Institute of the Pennsylvania Hospital; Neurologist, Pennsylvania, Philadelphia General and Germantown Hospitals. 111 N. Forty-ninth St.

1923. Stroud, William D., B.S., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Cardiologist to the Pennsylvania Hospital and Chief of Heart Clinics; Physician in Charge of the Children's Heart Hospital of Philadelphia; Cardiologist to Bryn Mawr Hospital. 1011 Clinton St.

1927. Sturgis, Samuel B., M.D. Ardmore, Pa.

1930. Sunderman, F. William, B.S., M.D., M.S., Ph.D., Assistant Professor, Research Medicine, University of Pennsylvania; Assistant Physician and Chemist, Pennsylvania Hospital; Chief of the Diabetic Clinic, Pennsylvania Hospital. 2015 Spruce St.

*1900. Swan, John M., M.D., Attending Physician, Park Avenue Hospital. 457 Park Avenue, Rochester, N. Y.

ELECTED

1928. SWARTLEY, WILLIAM B., M.D., Surgeon to Germantown Dispensary and Hospital and to Chestnut Hill Hospital; Demonstrator of Anatomy, Jefferson Medical College. 6002 Greene St., Germantown.

1900. TALLEY, JAMES ELY, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Presbyterian Hospital. 1811 Spruce St.

1931. TAYLOR, NORMAN H., A.B., M.A., M.D., Associate Pediatrist, Presbyterian Hospital; Assistant Pediatrist, Chestnut Hill Hospital. 100 W. Evergreen Ave.

1887. TAYLOR, WILLIAM J., M.D., Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes', Woman's and Abington Memorial Hospitals. 1825 Pine St.

1928. THOMAS, J. EARL, M.D. Drexel Hill, Pa.

1907. THOMAS, THOMAS TURNER, M.D., Chief Surgeon, Northeastern Hospital. 1737 Chestnut St.

1927. THORINGTON, J. MONROE, B.S., A.M., M.D., Instructor in Ophthalmology, University of Pennsylvania; Assistant in Ophthalmology, Presbyterian Hospital. 2031 Chestnut St.

1898. THORNTON, EDWARD Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1930. THORPE, EDWARD S., JR., B.S., M.D., Associate in Pediatrics and Assistant to the Dean, School of Medicine, University of Pennsylvania; Visiting Physician to Children's, University and Philadelphia General Hospitals. 133 S. Thirty-sixth St.

1912. TORREY, ROBERT G., M.D., Professor of Principles and Practice of Medicine, Woman's Medical College; Associate in Medicine, School of Medicine, University of Pennsylvania; Physician to the Philadelphia General Hospital. 1716 Locust St.

1908. TRACY, STEPHEN E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1930 Chestnut St.

1926. TUCKER, GABRIEL, M.D., Associate Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, and Associate Bronchoscopic Surgeon, University Hospital, University of Pennsylvania; Associate in Bronchoscopy and Assistant Bronchoscopist, Jefferson Medical College and Hospital; Bronchoscopist, Misericordia and Bryn Mawr Hospitals. 326 S. Nineteenth St.

ELECTED

†1894. Tunis, Joseph Price, M.D. University Club, Philadelphia.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. 1731 Pine St.

1929. Tyson, Ralph M., M.D., Professor of Pediatrics, Temple University School of Medicine. 334 S. Twenty-first St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-Patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Blairstown, N. J.

1873. Van Harlingen, Arthur, Ph.B., M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. Bryn Mawr, Pa.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

1912. Vaux, Norris Wistar, M.D., Clinical Professor of Obstetrics, Jefferson Medical College; Chief of Maternity Service, Philadelphia Lying-in and Pennsylvania Hospitals; Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 2031 Locust St.

*1897. Veasey, Clarence A., A.M., M.D., Ophthalmic Surgeon to the Deaconess and to St. Luke's Hospitals, Spokane. Suite 1569, Paulsen Medical and Dental Building, Spokane, Wash.

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1927. Wagers, Arthur J., M.D., Associate in Otology, Jefferson Medical College; Demonstrator of Laryngology, Jefferson Medical College; Assistant in Laryngology, Jefferson Hospital; Otologist, Joseph Price Hospital. 1429 Spruce St.

1928. Wagoner, George W., M.D., Assistant Professor of Orthopedic Surgery, Graduate School of Medicine, and Associate in Pathology, School of Medicine, University of Pennsylvania; Clinical Surgeon, Orthopedic Hospital; Orthopedic Surgeon, Bryn Mawr Hospital. Haverford, Pa.

1931. Walker, Herschel C., M.D., Assistant Surgeon, University Hospital; Instructor in Urology, School of Medicine, Univer-

ELECTED

sity of Pennsylvania and Woman's Medical College. 1323 Medical Arts Building.

1906. WALKER, JOHN K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-Patient Department of the Pennsylvania Hospital. Media, Pa.

1907. WALKER, WARREN, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1932. WALKLING, ADOLPH A., M.D., Assistant Surgeon, Pennsylvania Hospital; Chief of Surgical Out-Patient Department, Division B., Jefferson Hospital; Demonstrator of Fracture Dressings, Jefferson Medical College. Medical Arts Building.

1904. WALSH, JOSEPH, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium. Medical Arts Building.

1895. WATSON, ARTHUR W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

1923. WATSON, WILLIAM R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Chief Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1932. WATT, CHARLES C., JR., M.D., One of the Chiefs of Service in Medicine, Germantown Hospital; Assistant Visiting Physician, Orthopedic Hospital. 6605 Wayne Ave.

1903. WEBER, CHARLES H., M.D., Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Medical Dispensary, Children's Hospital; Pediatrist to the Municipal Court. 2048 Pine St.

1930. WEEDER, S. DANA, M.D., Assistant Surgeon, Germantown and Chestnut Hill Hospitals; Instructor in Anatomy, School of Medicine, University of Pennsylvania. 6110 Greene St., Germantown.

1920. WEIDMAN, FRED. D., M.D., Professor of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden; Attending Dermatologist, Philadelphia General Hospital. 20 Tenby Road, Llanerch, Pa.

1906. WEISENBURG, THEODORE H., M.D., Professor of Neuro-Psychiatry, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital, and Physician to Orthopedic Hospital and Infirmary for Nervous Diseases. 1930 Chestnut St.

ELECTED

1923. Weiss, Edward, M.D., Clinical Professor of Medicine, Temple University; Physician to the Tuberculosis Wards, Philadelphia General Hospital. 1923 Spruce St.

1913. Wells, P. Frailey, A.M., M.D., Ph.D. 754 N. Fortieth St.

1893. Westcott, Thompson S., M.D., Consulting Pediatrist to the Methodist Episcopal Hospital; Consulting Pediatrist, Haddock Memorial Home. 1720 Pine St.

1921. Whitaker, William, M.D. 5448 Germantown Ave.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia; Associate Professor of Bacteriology in the Graduate School of Medicine, University of Pennsylvania. 6611 N. Tenth St.

‡1905. Whiteway, Harold M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. Whiting, Albert D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 333 S. Eighteenth St.

1925. Widmann, Bernard P., M.D., Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Chief of the Radiological Department of the Philadelphia General Hospital. 250 S. Eighteenth St.

1914. Willard, de Forest P., B.S., M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Graduate, Orthopedic and Delaware Hospitals. 1916 Spruce St.

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1924. Williams, Horace James, M.D., Otolaryngologist to Germantown, Roxborough Memorial and Municipal Hospitals; Instructor in Otolaryngology, School of Medicine, University of Pennsylvania. 5908 Greene St., Germantown.

1920. Williams, Philip F., Ph.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Gynecologist and Obstetrician to Philadelphia and Jewish Hospitals; Obstetrician, Abington Memorial Hospital. 2206 Locust St.

ELECTED

1927. Williamson, Ernest G., M.D., F.R.C.S. (Edin.), Instructor in Clinical Surgery, Jefferson Hospital; Associate Surgeon, Children's Hospital; Assistant Surgeon, Presbyterian, and Clinical Assistant, Jefferson Hospitals. 6353 Woodbine Ave.

1916. Wilmer, Harry B., M.D., Assistant Professor of Clinical Allergy in the Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Germantown Hospital; Associate Physician, Presbyterian Hospital; Director of the Allergic Clinic, Germantown and Presbyterian Hospitals. 6013 Greene St., Germantown.

1921. Wilson, George, M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Clinical Professor of Neurology, Women's Medical College of Pennsylvania; Neurologist, Philadelphia General, Episcopal and Abington Memorial Hospitals; Assistant Neurologist, University Hospital. 133 S. Thirty-sixth St.

1874. Wilson, James Cornelius, A.M., M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief (Emeritus) to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 1930 Chestnut St.

1897. Wilson, W. Reynolds, M.D. Villa Nova, Pa.

1904. Wister, James W., M.D., Physician to the Out-Patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. 21 North Grand Ave., Baldwin, N. Y.

1918. Wolferth, Charles Christian, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Director of the Clinic, Robinette Foundation, University Hospital; Consulting Cardiologist, Jewish Hospital. S. E. Cor. Thirty-sixth and Spruce Sts.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General and St. Timothy's Hospitals. 2035 Walnut St.

1932. Wood, Francis C., A.B., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Fellow of the Robinette Foundation, University Hospital. Wynnewood, Pa.

1900. Wood, George B., M.D., Oto-laryngologist, Orthopedic Hospital; Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. N. E. Cor. Twentieth and Chestnut Sts.

ELECTED

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Philadelphia College of Pharmacy and Science. 319 S. Forty-first St.

*1911. Woods, Andrew H., A.B., M.D., Professor of Psychiatry, College of Medicine, and Director of the Psychopathic Hospital, University of Iowa. 1100 N. Dubuque St., Iowa City, Iowa.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. Worden, Charles B., A.M., M.D. Princeton, N. J.

1928. Wright, Carroll S., B.Sc., M.D., Professor of Dermatology-Syphilology, Temple University School of Medicine; Associate Professor of Dermatology-Syphilology, Graduate School of Medicine, University of Pennsylvania; Attending Radiologist, Philadelphia General Hospital; Attending Dermatologist, Municipal Hospital. 1402 Spruce St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon to the Wills Hospital; Consulting Ophthalmologist to the Glen Mills School and to St. Mary's Hospital. 1930 Chestnut St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

1931. Zozaya, José, B.S., M.D., Dr.P.H., Assistant Director of Mulford Biological Laboratories. Devon, Pa.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

‡1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., Cleveland Clinic, Euclid Ave. at E. Ninety-third St., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1916. Estes, William Lawrence, M.D., 805 Delaware Ave., Bethlehem, Pa.

1931. Garrison, Fielding H., M.D., 1900 E. Monument St., Baltimore, Md.

1912. McCaw, Brig.-Gen. Walter D., M.D., Assistant Surgeon-General, U. S. A., retired, Woodstock, Ulster Co., N. Y.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.

1906. Pilcher, Lewis Stephen, M.D., 386 Park St., Upper Montclair, N. J.

1892. Welch, William H., M.D. 1900 E. Monument St., Baltimore, Maryland.

Foreign

1928. Archibald, Edward, M.D., McGill University, Montreal, Canada.

1924. Hopkins, Sir F. Gowland, M.D., Biochemical Department, University of Cambridge, Cambridge, England.

1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., 317 Piccadilly St., London, Ontario.

1922. McCarrison, Colonel Robert, M.D., C.I.E., D.Sc. LL.D., F.R.C.P. (London), Honorary Physician to the King. Pasteur Institute, Coonoor, S. India.

ELECTED

1924. Macleod, John James R., M.D., M.B., D.Sc. (Hon.), LL.D., F.R.S., Marischal College, Aberdeen, Scotland.

1906. Myles, Sir Thomas, M.D., 33 Merion Square, W. Dublin, Ireland.

1885. Rendu, Jean, M.D., Lyons, France.

1928. Rolleston, Sir Humphry, Bart., G.C.V.O., K.C.B., M.D., D.C.L. (Durh.), LL.D., D.G.C., "Southfield," Trumpington Road, Cambridge, England.

‡1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., Ross Institute and Hospital for Tropical Diseases, Putney Heath, London, S. W. 15, England.

NECROLOGICAL LIST

FELLOWS

G. VICTOR JANVIER, M.D.	December 17, 1931
RICHARD S. HOOKER, M.D.	December 25, 1931
THOMAS C. ELY, M.D.	December 26, 1931
B. ALEXANDER RANDALL, M.D.	January 4, 1932
GEORGE C. STOUT, M.D.	January 27, 1932
FRANCIS B. JACOBS, M.D.	February 11, 1932
CHARLES M. MONTGOMERY, M.D.	March 3, 1932
JAMES HENDRIE LLOYD, M.D.	March 14, 1932
ELMER H. FUNK, M.D.	May 13, 1932
THOMAS A. COPE, M.D.	May 27, 1932
ORLANDO H. PETTY, M.D.	June 2, 1932
HAROLD M. WHITEWAY, M.D.	June 6, 1932
WILLIAM W. KEEN, M.D.	June 7, 1932
JOSEPH LEIDY, M.D.	July 6, 1932
SIR RONALD ROSS, M.D.	September 16, 1932
ASTLEY P. C. ASHHURST, M.D.	September 19, 1932
FRANK BILLINGS, M.D.	September 20, 1932
MAX H. BOCHROCH, M.D.	November 27, 1932
GEORGE FETTEROLF, M.D.	December 29, 1932

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

NOTE.—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York.
1923	Edward P. Heller, M.D.	Kansas City, Mo.
1924	Gordon Cameron, M.B., B.S.	Victoria, Australia.
1925	Raphael Isaacs, M.D.	Boston.
1926	P. S. Pelouze, M.D. and Frederick S. Schofield, M.D.	Philadelphia.
1927	Emil Bogen, M.D.	Cincinnati, Ohio.
1928	J. Rodman Paul, M.D. and William U. McClenahan	Philadelphia.
1929	George M. Dorrance, M.D.	Philadelphia.
1930	Henry A. Harris, M.D.	London (Eng.).
1931	Edgar S. J. King, M.D.	Melbourne (Australia).

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.
1924	George J. Heuer, M.D.	Cincinnati.
1926	Knud Faber, M.D.	Copenhagen.
1928	John James R. Macleod	Toronto, Can.
1929	Simon Flexner, M.D.	New York.
1930	Franz Alexander	Berlin, Germany.
1932	R. Tait McKenzie, M.D.	Philadelphia.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N. Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D., and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Macleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).
Mar. 5, 1924	Prof. A. Biedl	Prague (Czecho-Slovakia).
April 15, 1924	J. G. FitzGerald, M.D.	Toronto.
Oct. 31, 1924	John I. Hunter, M.D., and N. D. Royle, M.D.	Sydney (Australia).
Dec. 4, 1924	Willem Einthoven, M.D.	Leyden (Holland).
April 21, 1925	A. N. Richards, A.M., Ph.D.	Philadelphia.
Feb. 2, 1927	Eugene L. Opie, M.D.	Philadelphia.
Nov. 2, 1927	George R. Minot	Boston.
Mar. 22, 1928	Sir Humphry Rolleston, Bart., M.D.	Cambridge (Eng.).
Nov. 7, 1928	Ralph Pemberton, M.D.	Philadelphia.
Oct. 2, 1929	Joseph Barcroft, C.B.E., F.R.S.	Cambridge, (Eng.).
April 22, 1930	Sir Wilfred Grenfell, K.C.M.G., M.D.	Labrador.
May 7, 1930	Charles Singer, M.D., F.R.C.P.	London (Eng.).
Nov. 5, 1930	Prof. M. Weinberg	Paris (France).
Feb. 4, 1931	W. J. Merle Scott, M.D.	Rochester, N. Y.
Mar. 4, 1931	George Burgess Magrath, M.D.	Boston.
Oct. 28, 1931	Sir Thomas Lewis, M.D.	London (Eng.).
Nov. 4, 1931	F. J. Lang, M.D.	Innsbruck (Austria).

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.

1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M.D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D.	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Prof. H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.
1924	Evarts Graham, M.D.	St. Louis.
1925	Edward Archibald, M.D.	Montreal, Can.
1926	Walton Martin, M.D.	Ithaca, N. Y.
1927	E. Starr Judd, M.D.	Rochester, Minn.
1928	Herbert W. Carson, M.D.	London (Eng.).
1929	Robert B. Greenough, M.D.	Boston.
1930	Frank H. Lahey, M.D.	Boston.
1931	Edwin Beer, M.D.	New York.
1932	H. Gideon Wells, M.D.	Chicago.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D.	New York.
1925	Simon Flexner, M.D.	New York.
1928	Frederick Tilney, M.D.	New York.
1931	Francis R. Packard, M.D.	Philadelphia.

MEMOIR OF DR. FRANCIS X. DERCUM*

By CHARLES W. BURR, M.D.

Francis Xavier Dercum, son of Ernest and Susanna (Erhart) Dercum, was born in Philadelphia, August 10, 1856. He came of good stock. In the troubled days of Henry VIII, the family were living in Cumberland County, England, but, being ardent Catholics and siding with the pope in the religious controversies, soon migrated to the Netherlands. In 1689 Peter von Dercum was the Administrator of the ruling house at Adendorf, to which office his grandson, Laurenz Anton von Dercum who married Maria Theresia, Countess von Ravelsberg, succeeded him. Dercum's great-grandfather, Franz Karl Joseph Johann von Nepomuk von Dercum, was born at the Castle Adendorf in Eifel and studied law at Koblenz and Heidelberg. Later he was appointed to the Chancery Court and became director of the Archives. Dercum's grandfather was graduated at Metz, then a French city, studied at St. Cyr, the military school, and later became an officer in the French Army. He was with Napoleon at Waterloo and, after the destruction of the Empire, went to the Palatinate, entering the bureaucratic service, first at Contwig and later at Zweibrücken. He came to America in 1849. There were men of science, law and learning in the family, generation after generation. Dercum's father, Ernest, was born at Contwig, July 15, 1825. He was imbued with French ideas of "Liberty, Equality, Fraternity," and wrote political pamphlets against the government, like many others in that time of Revolution. Europe was seething with social unrest. The liberals in Germany had dreams about the rights of man. They talked and fought and lost. Reaction conquered, and Dercum's father, like many others, fled to America, the land of their dreams, in 1848. This was the period of a quite large migration from Germany of men who became very valuable citizens. They, Carl Schurz was one of them, were inspired

*Read, January 11, 1932.

by no low motive, but yearned to live in a land, free politically, intellectually, socially, and, their efforts failing at home, they came here. Ernest Dercum established a book-importing business in Philadelphia and later became a grain commission merchant.

Dercum's uncle, Herman, who also came to America, organized an artillery company of which he became captain, at the outbreak of the Civil War. He was in charge of Fort Ethan Allen, and died as the result of injuries received when it was blown up.

Dercum attended the public schools of Philadelphia, graduated from the Central High School, and, entering the medical department of the University of Pennsylvania, received the degree of M.D. therefrom in 1877. Among his classmates were Drs. James M. Anders, John H. Musser, George A. Piersol, A. J. Parker, Thomas H. Fenton, Henry Formad and Joseph Price. Several of these men were later teachers in the medical school of their Alma Mater. All are dead save Dr. Anders, who, as all of us know, is very much alive, active in medical matters, especially public health, and professor emeritus in the postgraduate school. A. J. Parker, who, in my undergraduate days, taught zoölogy to the students in the College preparing for medicine, was intellectually brilliant and almost a genius, but was born with the peculiarities of temperament often associated with, but not causative of genius, and died while still a young man. He and Dercum had much in common intellectually and did some scientific work together. It was a noteworthy class. Later Dercum was given the Ph.D. degree.

From schoolboy days, he showed an inborn interest in natural history (the word biology was only beginning to be used) and early joined the Academy of Natural Sciences. From student days he was greatly influenced by Joseph Leidy and studied human anatomy, not only as a subject necessary to the physician, but as a center around which to group the facts of comparative anatomy. It was a period of great activity in the natural sciences and the height of the quarrel about evolution between men of science and theologians. All his teachers in the natural sciences accepted Darwinism, and thus planted in him very early acceptance of the idea of evolution, which he retained through life, though his opinion as to its methods changed with increasing knowledge.

His first papers were not medical but biological and were printed in the *American Naturalist* and the *Proceedings of the Academy of Natural Sciences*. They were "On the Sensory Organs, Suggestions

with a view to Generalization" (1878) and "On the Morphology of the Semicircular Canals" (1879). Throughout life he never lost interest in biology and his philosophy was based on it. His first teaching position was demonstrator in the laboratory of Physiology under Professor Harrison Allen.

Like all the men of his generation who became specialists, he began as, and continued to be for quite a few years, a general practitioner. But by 1884 his interest in neurology had become so great that he, with Wharton Sinkler and J. T. Eshridge, joined Dr. Charles K. Mills, who was the originator of the movement, in founding the Philadelphia Neurological Society, S. Weir Mitchell being the first president. He later was president and until death an active member. Cerebral localization, and especially aphasia, were for years the most important subjects brought before the society. The discussions were often fast and furious; sometimes speakers were a little too dogmatic, and on one occasion a member, usually very dignified, even announced he would "eat his hat if something were not true." In recent years a more scientific calm has usually pervaded the meetings. I, who am a little, alas only a little, younger than Dercum, joined the society later, and, I am almost ashamed, in these watery days, to confess it, enjoyed the beer at a nearby hotel and the talk after the meetings quite as much as the pure science. But we have changed all that.

I shall not read the long list of titles of Dercum's papers, but wish to refer to a few. In the eighties the causation of convulsions, especially of those not associated with organic disease, was being widely discussed, and Dercum, with Andrew J. Parker, in 1884, made some interesting experiments "On the Artificial Production of Convulsive Seizures." They found, for example, clonic convulsions could be produced by the muscular constraint ensuing from holding the outstretched arm just not in contact with the table. Like myself, in those early days, he studied hypnotism very seriously; but, like myself, he became discouraged, because it is so saturated with fraud and self deception, and so much depends on the honesty of the person used in the tests. Hypnotism is immortal, or rather it dies and is reborn repeatedly under another name. This has been going on for centuries. It always flourishes in times of social unrest and intellectual upheaval. Mesmerism, in France in Franklin's time, is an example of the former; its use in psychoanalysis, in the beginning of the popularity of that form of treatment, of the latter.

In 1884 he was appointed the first consultant pathologist to the State Hospital for the Insane at Norristown, making the first annual report in 1885. I think this establishment of a department of pathology was the first instance, in Pennsylvania, of the managers of a state hospital granting any opportunity for scientific work beyond clinical study. The rule did not pass without great opposition on the part of a few members of the board, who, being themselves ignorant and intellectually moronic, made the then usual stock argument that the doctors would next want to experiment on the living. Also, these lovers of the "dear peepul," were not going to allow their much-loved constituents to be cut up when dead.

Dercum was associated with Muybridge in making a series of photographs of moving animals. This work of Muybridge was the beginning of what has developed into "the movies."

In 1884 he became chief of the nervous clinic of Dr. Horatio C. Wood in the University of Pennsylvania, in succession to Dr. Charles K. Mills, and it was then that I first became acquainted with him, though it was quite a few years before we began to know each other well. Many students attended Wood's clinics, attracted quite as much, I suspect, by the dry humor and caustic wit of Wood, and the melodramatic performances of some patients, as by the desire to learn. But Wood stimulated all of us. Dercum, ambitious, young and, therefore, unsure, and fearing a little the criticism of ingenuous youth, held the clinic on the rare occasions of Wood's absence. Thus he learned how to teach.

In 1892 he married Elizabeth DeHaven Comly, a member of an old Philadelphia family, the granddaughter of Comly's *Spelling Book*. In my boyhood days it was still popular with schoolmasters and unpopular with small boys, born minus the natural ability to spell. Mrs. Dercum was, through all the years of their married life, an ideal helpmate and mother to their children.

In 1893 he was elected to the newly-created chair of clinical professor of neurology at the Jefferson Medical College. In 1900 he was promoted to a full professorship, and continued in it until 1925 when he became professor emeritus, being succeeded in his professorship by Dr. Edward A. Strecker.

From the eighties on, there had been increasing interest in the study of the endocrine glands and in the causes of growth and development of the different systems and organs of the body. In consequence, physicians were on the lookout for unusual appear-

ances in surface anatomy, and, in 1892, Dercum published his extremely important article on "Adiposis Dolorosa," now frequently called Dercum's disease.

During his life he wrote many papers and several books. In 1895 he was editor of and contributor to *A Text-book of Nervous Diseases by American Authors*, the first American book on nervous diseases of multiple authorship. He wrote also on *Rest, Suggestion and Other Therapeutic Measures in Nervous and Mental Diseases*, *A Clinical Manual of Mental Disease*, *Hysteria and Accident Compensation* (1916), *The Biology of the Internal Secretions* (1924), and *The Physiology of the Mind* (1925). As time passed, he became more and more interested in philosophical and psychological matters and wrote a little on these subjects. His industry, ambition and mental alertness are shown by the fact that he wrote so much while burdened with the cares of a large practice. His most important patient, from the public point of view, was President Wilson, whom he attended in 1919 and 1920.

In 1927, he was elected president of the American Philosophical Society, which he had joined thirty-five years before, and continued in office until his death. His last papers were read before it. Two were "On the Nature of Thought and Its Limitation" (1930) and "Non-living and Living Matter" (1931). Before his death, he had proposed, and started on its way, an investigation by the society of the present state of human knowledge, an intellectual stocktaking. This, when completed, will be a tremendously important piece of work.

He was a member of many societies: President of the American Neurological Association, of the Philadelphia Neurological and Philadelphia Psychiatric. He was elected to fellowship in this College in 1885, and a member of its library committee and chairman many years. He was foreign corresponding member of the Neurological Society of Paris, corresponding member of the Psychiatric and Neurological Society of London, the Royal Medical Society of Buda-Pest and of the Neurological Section of the Royal Society of Medicine of London. He was a very active member of the state and county societies and of the American Medical Association.

A man is what environment, acting on the stuff he is made of, makes him. The stuff depends on his ancestry. The environment, in infancy and childhood, so far as he is concerned, is accidental, but may be controlled, for good or evil, by others. Dercum's

ancestry was excellent, the environment of his childhood wise. He was the happy product of mid-Victorian days, and the pattern of his mind was formed and fixed before the onset of the present great, interesting and revolutionary overturning of all things—political, scientific and religious.

His earliest special interest in medicine was in organic neurology and physiology; interest in psychiatry came later. This was fortunate, because, when a very young man becomes intensely interested in the study of mental function, he is prone to become absorbed in speculation and metaphysics, and loses the strict mental discipline of physiology and anatomy. To theorize is so easy, to reason so difficult. When Freudian psychology came upon the scene, he found it impossible to accept it, because it required the assumption of so many unproved and unprovable things—dream symbolism, the subconscious, the censor—and because it laid so much stress on *libido*, which Freud used in no sentimental sense, but with a very crude, positive and physical meaning. Of course, the psychoanalysts answered that he was so crystallized in mind, he could not accept new and great discoveries. Years later, he accepted the new and startling, but seemingly proven, ideas in physics. The younger and most recent disciples of the cult of Freudism have softened and poetized the meaning of *libido*.

He was a materialist, but quite as emotional and dogmatic as the idealists. He denied design and purpose in the making of the Universe, could not accept God either as artist or mathematician, and believed accident and chance have made things as they are. If Democritus, who flourished about 410 B.C., could revisit the glimpses of the moon, he would naturally be interested in current philosophy, and the ideas of men like Dercum would sound strangely familiar to him; he might even think the moderns had not progressed very far. Or, he might be somewhat flattered when he learned from many scholars that "Modern science largely busies itself with proving the accuracy of the guesses of Greek genius." Dercum was not a rationalist in the proper sense of the word—a man who has no emotional longing to believe anything, but simply desires to find truth, careless and unconcerned whither it leads, and accepting the fact that human intelligence cannot know things in themselves but only as they react on him. He had a very emotional desire to believe and disbelieve. He would show marked feeling when some of his friends, from an impish desire to start an argument, would

pretend to philosophical opinions he opposed and they really were in grave doubt about or did not believe at all, and never rose to the height of that great argument—we know not anything but only seeming. He was as naïve in his attitude toward Berkleyian idealism as that old Tory, Dr. Johnson.

The manner of his death was ideal; sudden, and while in actual performance of duty. He was presiding at the meeting of the American Philosophical Society, in its building in Independence Square, on April 24, 1931. He was sitting in the chair used by the president since Franklin's time; his head dropped forward; he sank in collapse; his heart stopped; eternal silence came.

He was a very human man, with strong emotions; a good friend, a not-too-violent enemy. He did not hate bitterly, nor cherish bad feeling. He was very fond of music, an inheritance from his mother.

He was a skilled physician, an instructive writer, an excellent teacher and he did his task in life and enjoyed the doing until the end.

MEMOIR OF HOBART AMORY HARE, M.D.*

By THOMAS McCRAE, M.D.

With the passing of Hobart Amory Hare, medicine lost an eminent practitioner and a wise consultant, Jefferson Medical College a celebrated member of the Faculty and this College a former President and one who had long taken a part in its affairs. A man of unusual ability and with remarkable energy, he attained eminence as a practitioner, teacher and author. Probably he had instructed more students than any teacher in this country. It may be difficult to decide in which of his many interests a man takes the greatest pleasure. With Dr. Hare, it is probable that teaching took first place. Not long before his death, in a discussion as to what had given the greatest satisfaction in life, his statement regarding himself was that he had this feeling when he knew that he was giving a good lecture or clinic. He felt very strongly the great importance of training students properly in the care of the sick. His views on the need of the thorough teaching of therapeutics were strongly held and he resented as insufficient certain instruction given under that designation. His teaching was incisive and dogmatic and illustrated by a wealth of description and apt comparison. As a consultant he was able to draw on a store of valuable experience.

Dr. Hare was born in Philadelphia, September 20, 1862, the only child of Bishop William Hobart Hare, of South Dakota, and Mary Amory Howe Hare. There was a strong ecclesiastical strain in his ancestry, as his maternal grandfather was Bishop Mark Antony DeWolfe Howe. It seems probable that his ancestry endowed him with the ability to write.

His preparatory education was received at the Episcopal Academy of Philadelphia, after which he entered the University of Pennsylvania and graduated in medicine in 1884. He received the degree of B.S. in 1885. Soon after this he went abroad for a period of study, and perhaps the most noteworthy work done during this time was at Leipsic, where he worked under Ludwig, chiefly in

* Read January 11, 1932.

physiological research. Undoubtedly this particular contact had much to do with his subsequent career. It introduced him to research work and perhaps was largely responsible for the unusual amount of work which he produced in a comparatively few years after his graduation. He also worked in Berne and in London, but the men with whom he was in contact in these places do not seem to have had the same influence on his subsequent career.

Soon after his return to Philadelphia, he was appointed lecturer in physiology at the University of Pennsylvania. This proved to be a fruitful period in research work, especially that concerned with the action of drugs. In the few years following graduation he carried off an unusual number of prizes. On his graduation he was awarded the Faculty Prize for his thesis; he was twice the recipient of the Fiske Fund Prize of the Rhode Island Medical Society, in 1885 and 1886; the Cartwright Prize of the New York College of Physicians and Surgeons in 1889 (with Dr. Martin); the Warren Triennial Prize of the Massachusetts General Hospital in 1889 (with Dr. Martin); the prize offered by the Royal Academy of Belgium for the best essay on epilepsy in 1889; and the Boylston Prize of Harvard University in 1890. The greatest honor was the award of the Fothergillian Gold Medal of the Medical Society of London in 1888 for his work on "Mediastinal Tumors." He was the only American who has ever obtained this distinction. This medal he bequeathed to this College. I remember very well one of the older generation in Baltimore telling me that, in the late eighties, medical men were asking who this young man in Philadelphia was who seemed to be carrying off all the prizes in the country.

That he had "arrived" at an earlier age than is usual was shown by his election, in 1890, to the position of clinical professor of diseases of children at the University of Pennsylvania. It may be that those responsible for this and his next appointment had acumen more acute than is usual in picking a coming man. In the following year (1891) he was called to the chair of therapeutics and materia media in the Jefferson Medical College. This position he filled for forty years, an unusual term of service. His teaching was clear and emphatic and illustrated by a wealth of illustration and anecdote. He held very strongly the importance of training students in what should be done in the proper care of the sick. The teaching of methods of treatment which were concerned more with theory

than practice aroused his criticism. Reference should be made to one procedure in treatment in which he had great skill, the treatment of thoracic aneurysm by wiring and electrolysis. He did this exceedingly well and with great success in a number of cases. One important piece of work was that done as a member of the Hyderabad Chloroform Commission in 1894. As a token of appreciation he was presented with a piece of plate, which he bequeathed to this College.

He was engaged in medical journalism throughout the greater part of his life. His first efforts were as editor of *The University Medical Magazine*. Later he was editor of *The Medical News* for a time and subsequently of *The Therapeutic Gazette*. He also edited *Progressive Medicine*, a quarterly digest, for thirty-two years. As an author he was unusually prolific. His work on *Mediastinal Disease* was a thorough study of the material available at the time. It has been a most useful work to those interested in intrathoracic tumors. His greatest success was the textbook on treatment, *Practical Therapeutics*, which went through twenty-one editions. It is one of the most widely used books on therapeutics ever written. His work on *Symptoms in the Diagnosis of Disease* went through nine editions and *A Textbook of the Practice of Medicine* had three editions. He edited a system of treatment in two volumes, *Modern Treatment*. A monograph on the *Medical Complications and Sequelæ of Typhoid Fever and the Other Exanthemata* went through two editions. One can only wonder at the industry which made possible so great a literary output. In addition, he wrote many journal articles on a wide variety of subjects.

Of the honors that came to him, probably the one in which he felt the greatest satisfaction was the position of President of The College of Physicians, which he held from 1925–1928. He was elected a Fellow of this College in 1889 and a member of the Association of American Physicians in 1890. He received the honorary degree of LL.D. from the University of Pennsylvania in 1921.

One noteworthy activity was his membership on the Board of City Trusts. This demanded an expenditure of much time and energy. He was keenly interested in the work of Girard College and, naturally, his advice in its medical and health problems was of the greatest value. He wrote an interesting account of Stephen Girard and his work in the yellow fever epidemic.[1] From some of

[1] Ann. of Med. History, 1930, n. s. 2, 372.

his colleagues on this Board it was learned that he showed unusually keen business sense and judgment. One colleague said that a splendid business man was lost when Hobart Hare decided to follow medicine.

As to his personal qualities, one might say that he did not suffer fools gladly. His mind worked so rapidly that he was sometimes impatient with the slower mental processes of the average man. Sometimes he might be quick and almost brusque in his manner. This undoubtedly was due in great part to the fact that he suffered from a recurring headache, which, for want of a better name, might be termed migraine. He told me that it was a rare day that passed without an attack of severe pain for a varying period. It might be for half an hour or for the greater part of the day. I have seen him just before a lecture or clinic sitting with his hands pressed on his head and groaning with pain, but no one present at the lecture or clinic would suspect from his manner how severely he was suffering. This was never allowed to interfere with his teaching, and he said very little about it. Curiously enough, in his last illness he was free of these attacks.

One hesitates to dwell too intimately on one's personal relationships with one who is gone, yet these may give a clearer idea of what the man was. Coming to Philadelphia, as I did, a comparative stranger, he need not have felt any compulsion to go out of his way to help a newcomer, but had he been my older brother he could not have been kinder or more helpful to me. Perhaps the majority of us did not think of him as an "outgoing" man. He was not one who wore his heart on his sleeve. With such a man, as a rule, it is only after he has gone that we learn of kindly deeds done and of the depth of personal affection felt for him. Such was the case with Hobart Hare.

He married Miss Rebecca Clifford Pemberton, of Philadelphia, in May, 1884. They had one daughter, Mrs. James P. Hutchinson, who has inherited the literary ability of her forebears.

His death occurred on June 15, 1931, after an illness of six months in which he endured severe pain with stoical philosophy and faced the inevitable end with courage.

Of Hobart Hare we may say that in him was a rare combination of abilities as a physician, teacher and author. He had a life full of work and achievements. He leaves behind him a record of usefulness and duty well done. What better can be said of any man?

JOHN MADISON TAYLOR.*

An Appreciation

By SOLOMON SOLIS-COHEN

The Secretary of the Princeton Class of 1876 writes of John Madison Taylor, that "he had a buoyant nature and an almost boyish enthusiasm, which he carried to the end of his life." This is written in connection with his skill and delight in athletic sports, and his employment of outdoor life in the rehabilitation of his patients; but it applies as well to his work in all fields. To that, let us add his overflowing vitality and his kindly and unsimulated interest in the individual under treatment, and we have accounted for a large portion of his success; not only with the nervous, the despondent, the sluggish, but also with those suffering from more tangible affections, and perhaps especially with children.

But these qualities, worthy though they are, do not give us a complete picture of Madison Taylor.

As a man, his outstanding characteristics were a high sense of honor, a genuine kindliness of spirit and an intense loyalty. He was jealous not only of his personal honor, but also of the honor of his profession; he was kind to his patients, rich and poor alike, and tolerant of their failings and their whims; he was loyal to ideals, loyal to principles, loyal to friends, loyal to intellectual and moral convictions in science and in life.

As a physician, Dr. Taylor showed a scientific inquisitiveness and openmindedness that made him into that delightful paradox, a radical conservative—or, if one prefer the colloquial terminology—a stand-pat progressive. Pope's cautious and somewhat grovelling counsel:

> "Be not the first by whom the new is tried
> Nor yet the last to put the old aside—"

he contemned and condemned, in both its branches.

He taught in the therapeutic field, and we may take that teaching as an illustration of his catholicity. The novelty of a suggestion

* Read January 11, 1932.

was, to him, no cause for charging its escutcheon with a bend sinister; nor, on the other hand, was the antiquity of a therapeutic practice a sufficient reason for its damnation. If the new seemed worthy—founded upon careful observation and sound reasoning—he welcomed it to trial. If trial proved its worth, he continued to use it; if not, he put it aside. If the old was tried and proved, he retained it in his armamentarium, to be used when needed. For both in his conservatism and in his radicalism, his standing-pat and his progressiveness, he discriminated—so that while he did in a way follow the epistolographer's precept to "prove all things and hold fast that which is good," the proving in many instances was intellectual merely. That only which passed the intellectual test was given the practical one; and only that which the practical test proved good, did he hold fast.

He used drugs when he thought them necessary, but he preferred physical measures, and those products of the animal organism itself, that we now term "hormones." He used mental influence legitimately and successfully.

An example of his progressiveness, his intellectual fearlessness and his independent but instructed thinking, was his attitude toward the therapeutic use of digital manipulations. These he advocated and employed for their effect upon the autonomic nerve system, and through it upon the organic functions—especially the circulation.

Perhaps his long association with Dr. Weir Mitchell, and with the work of the Orthopedic Hospital in massage, had fitted him to understand better than do most physicians, the actuality of such circulatory and organic reactions. At all events, he saw a potent method of influencing the bodily mechanism largely abandoned to charlatanry and ignorance; and he wished to rescue it for the service of disciplined medical art. He observed its workings and sought to establish its true status—its indications and its counter-indications, its powers and its limitations.

I have been under the impression that to him we owe the coinage of the word "*cheiropraxis*"—I need not here point out its Greek roots—to indicate a definite order of practice with the hands. He was not responsible, however, for its degradation into "chiropracty," or for the claims made by those who call themselves "chiropractics."

This is not the place to discuss the value of what, in my teaching, I have called "Taylor's methods;" or the still larger subject of the negligence by which physicians have allowed the measures, physical

and mental, of physiologic therapeutics, to fall so largely into unworthy or unskilled or unintelligent control. Enough to say that Madison Taylor was, for long years, one of the few to recognize the immense importance both of the traditional and the novel, among measures of this class; and one of the leaders in restoring them to their just standing in medicine. For this he was willing to risk the lack of sympathy, the scepticism, even the depreciation and ridicule, that is too often the reward of pathbreakers; as it is too commonly, also, the guerdon of them that would conserve and utilize ancient knowledge.

Madison Taylor was likewise one of the first to recognize the importance, in pathology, in diagnosis and in treatment, of the "ductless glands," and their "internal secretions"—(one may properly revive, in this connection, the terminology of those early days of doubt and scorn). With similar openmindedness, he was one of the few to realize—despite the general scepticism when the topic was first brought forward some forty years ago—the actuality and the moment of autonomic imbalance (or—as the condition was, at first, too narrowly termed—"vasomotor ataxia") as the constitutional basis of various and varying disorders, some of them highly important.

But these are only particular instances of his general attitude toward new truths.

A brief record may be made of his schooling, his professional appointments, his writings, and like data.

Biographical Data

John Madison Taylor was born in Lancaster, Pennsylvania, July 4, 1855. He was the son of Major William Johnson and Mary (Bearden) Taylor.

His father was an analytical chemist and mining engineer, who died of acute pneumonia contracted while serving as Major of the "Purnell Legion," a Maryland volunteer regiment, in April, 1864. His mother was a daughter of Marcus D. and Elizabeth (Cocke) Bearden, of Knoxville, Tennessee, and through her he was descended from William Cocke, one of the first two United States senators elected from Tennessee. The paternal ancestor was William Taylor, who emigrated from England to Virginia in 1634. And the subsequent genealogy is as follows:

William Taylor 2d, Elias, George, Mathias, Samuel, John, William Hazzard Taylor, whose wife was Eliza Johnson, and John Madison Taylor, who was the grandfather of Dr. Taylor.

Among other ancestors may be mentioned Sir William Aston, who emigrated from England to Virginia in 1664; besides prominent members of the Howell, Emlen and Johnson families in Pennsylvania, New Jersey and Maryland.

After studying at Rugby Academy, Philadelphia, Madison Taylor was graduated from the College of New Jersey, Princeton, in 1876, receiving a B.A. degree, and in 1879 his M.A. degree. He received the degree of M.D. at the University of Pennsylvania in 1878.

Madison Taylor was, in the course of his career, resident (1878–1879), and later, assistant physician (1880) in the Children's Hospital; chief of the children's clinic in the University Hospital (1879–1883); chief of the throat and chest clinics, also neurologist, at the Howard Hospital (1864–1901); professor of diseases of children at the Philadelphia Polyclinic and College for Graduates in Medicine (1885–1901); pediatrist to the Philadelphia General Hospital (1900); professor of diseases of children at the Medico-Chirurgical College (1903); assistant professor of applied therapeutics at Temple University Medical School (1910); later professor of pediatrics (1915), and still later, professor of physical therapeutics and dietetics (1918–1923) at the same institution.

He was consulting physician to the Elwyn Training School for Feeble-minded Children, the Vineland Training School for the Feeble-minded, and "The Lindens," in Haddonfield, New Jersey, a private school for backward children.

He was also one of the board of managers of the Philadelphia Skin and Cancer Hospital (1929–1931). He was personal assistant to Dr. S. Weir Mitchell, and chief of clinic to the latter at the Orthopedic Hospital (1882–1896).

From early boyhood Madison Taylor was much interested in draftsmanship and coloring, and filled many sketch books even while at school and college. He had the delightful habit of illustrating his letters with pen-and-ink sketches. He was naturally led to apply this ability to his profession, producing many illustrations, both in black and white and in color, not only for his own works, but for those of his friends. Thus he illustrated much of Dr. S. Weir Mitchell's later work on snake venom; Dr. W. W. Keen's operative findings in brain surgery; Dr. J. Solis-Cohen's study of pharyngeal and laryngeal tuberculosis.

With Dr. William H. Wells, he was joint-author of a *Manual of Diseases of Children* (1898–1901–1913). In 1914 this work was revised by McKee and Wells and the name changed to *Practical Pediatrics*. To this edition Dr. Taylor contributed an appendix on "Development and Deformation of Children."

He was associated as editor with Dr. Charles E. deM. Sajous in the latter's *Analytic Cyclopedia of Practical Medicine*.

Among his publications, which included more than three hundred and fifty essays in medical, scientific and lay journals, is a book, *Ourselves, a Personal and Family History Register*. This is a series of elaborate forms for recording the data of heredity and of individual health and disease from birth to death. To have such records of patients would prove extremely valuable. But since the ordinary lay person—or physician, for that matter—will not "take the trouble" to fill the blanks properly, it belongs to that "medicine of the future," of which Dr. Taylor wrote so entertainingly.

At the time of his death, Dr. Taylor was at work on a book on *Convalescence*.

Of his papers, the following titles illustrative of their wide range and philosophic trend may be cited:

"Biskra. An Ideal Winter Climate," "Urticaria of Pharynx," etc., "Exophthalmic Goiter," "Repair of Will-Loss," "The Heart and Circulation in the Feeble-minded," "Food and Work," "Conservation of Human Energy." "Beauty," "Sleep and its Regulation," etc., "Physiology as the Basis of Clinical Medicine," "Paroxysmal Neuroses," "Curative Factors Climatic to Normal Environment," "Internal Secretions in Senility," "Saline Solution in Pneumonia," "Future Science of Medicine," "Management of Exhaustion States in Men," "Potency of Suggestion," "Pathology of Crime," "Derangements of the Vasomotor Equilibrium," "Clinical Records of Families," "The Hand as a Therapeutic Agent," "The Curative Powers in Human Milk," "Attitude of the Medical Profession Toward Osteopathy," "Physician and Masseur," "Psychotherapy: What it is and what it is not," "Psychic Hypertension," "Orthotherapy. Normality of Posture as a Factor in Health," "Cardio-vascular Renal Regulation by other Means than Drugs," "Physico-dynamic Remedies," "Physico-dynamic Agencies in Diagnosis and Treatment," "Psycho-analysis," "Chronic Miseries of Outdoor Workers," "Training Children of Nursery Age," "Drugless Healing Cults," "Reduction Cures for

Overweight," "Reconstructive Therapeutics," "Premature Old Age," "Shall the Brain Worker Take Exercise?" "Fatigue and Exhaustion," "Is it Worth While for a Man of Fifty to get into Good Physical Condition?" "Types of Men Observed Among Recruits," "Belief and Error; their Effects on Behavior," "Tonic Spasm as a Cause of Disability and the Remedy therefor," "Tremor, with Special Reference to Obstinate Types," "Perils of Adulthood, Chiefly those of the Autumn of Life," "Rest and Work; Weariness and Recreation," "How the Middle-aged Man or Woman Ought to Feel," "Poise or Tranquility," "Periodicities," "Nervousness or Nervous Exhaustion," "Undefined but Commonly Recurring Distresses," "Calcium Metabolism," "Nervousness a Term to be Either Defined or Abandoned," "What to Do for Tuberculosis," "What May We Learn from M. Coue's Teaching?" "The Control of the Circulation by Simple Physical Methods," "The Redemption of Overwrought Leaders from Energy Leaks," "Repair of Hearts Through Performance of Movements," "Treatment of Certain Conditions Common to Most Chronic Diseases," "How Does Disease Begin?"

In his professional capacity, Dr. Taylor accompanied many prominent men while traveling or on hunting expeditions. He became an ardent sportsman and secured specimens of most of the American big game.

The summer of 1886 he spent with Lispenard Stewart on Theodore Roosevelt's ranch in Dakota. In 1891 he traveled abroad with Joseph Pulitzer, and during this summer and 1892 he was physician to James G. Blaine, who regained health under Dr. Taylor's care. As a student of nervous diseases and their resultant conditions of exhaustion, he was successful in treating many such cases by rational hygienics and out-of-door measures.

Dr. Taylor was married on October 15, 1879, to Emily, daughter of Dr. Henry Edward Drayton and Mary (Brady) Drayton. Mrs. Taylor is a well-known miniature painter, and is president of the Pennsylvania Society of Miniature Painters.

Dr. Taylor became a fellow of the College in 1886. He was likewise a fellow of: The American Academy of Medicine, The American Therapeutic Society, The American Climatological Society, The American Medical Association, The American Neurological Society. He was a member of: The Philadelphia County Medical Society, The Philadelphia Pathological Society, The Philadelphia

Neurological Society, The State Medical Society of Pennsylvania, and an Associate of the Société Hygiénique de France. He was also a member of many clubs and organizations: Among them, the Philadelphia Sketch Club, the Academy of Natural Sciences of Philadelphia (1879), the University Club (charter member), the Philadelphia Fencing and Sparring Club (V. P. 1882–1898), Southern Club, Princeton Club, Sons of the American Revolution; Loyal Legion (Companion), Philadelphia Country Club, Alpha Mu Pi Omega Medical Society, U. of P. Medical Alumni Association (Pres. 1900–1901). He was President of his medical class of 1878.

Dr. Taylor's active career was ended abruptly in his seventy-sixth year, when he died of uremic poisoning on October 3, 1931.

MEMOIR OF CHARLES HARROD VINTON, M.D.*

By BURTON CHANCE, M.D.

On August 20, 1931, there passed out of this life a Fellow of the College who, owing to ill-health, had retired from professional activities many years before, yet, who, because of the benefits which he had received from his Fellowship, had maintained his regard and affection for the College, and who at his death it was learned had bequeathed to it twelve thousand dollars, contingent upon the life interest of a designated person in his own intimate circle.

Charles Harrod Vinton was elected a Fellow, May 2, 1883. He had received his degree on March 13, 1868, after the usual course in the medical school of the University, then at Ninth and Chestnut Streets. His general education was obtained in the public schools of the city, the degrees in course having been conferred on him by the high school in 1865. Following his graduation he became associated at the Episcopal Hospital and in private practice with Dr. Albert Fricke, a distinguished physician in the community which at that period dignified the region about Franklin Square, at Sixth and Vine Streets. But, because of ill-health, these pleasant associations were soon broken. After an interval of several years, in 1876 he reëntered practice at 1617 Race Street, on the north side of the Friends' Meeting, where he continued to reside until his final retirement. For a number of years he served on the staff of Dr. Douglass Hall at Wills Hospital, and during this period his practice was confined to ophthalmology.

In 1885 Dr. Vinton married Miss Maria Conaroe, the daughter of Mr. George Conaroe, a well-known portrait painter. Mrs. Vinton died in 1906. There were no children by this marriage and Dr. Vinton withdrew himself more and more into private life, devoting himself to the many charities in which both he and his wife had been interested for many years.

Dr. Vinton was devoted to music and art and to literature. He was fluent in French, in Latin and in Greek, besides manifesting a

* Read January 11, 1932.

deep culture in English. He delighted to translate as well as edit poems and sonnets in the various languages, yet these exercises were for his own amusement and the delight of his intimate friends. Although he read extensively in the medical sciences, keeping himself abreast of the times and associating himself with his professional fellows, participating in the discussion at the meetings of the College, no record of any writings or of the publication of communications of a scientific nature by him has so far been found. By 1897, because of increasing physical debility, he retired from all active participation in practical affairs, yet he never lost interest in his profession and continued his Fellowship and his membership in a number of scientific societies with which he had been identified. On "Alumni Day" at the University, in June last, he represented the class of '68 Medical. His last years were spent quietly at Atlantic City, the summers at Ogunquit, Maine.

His sight began to fail when about in his seventieth year, through cataract; but by the skilful operation on one of his eyes by Dr. Hansell, happily his sight was restored in 1922, when he was in his seventy-seventh year. He pursued his quiet life until, after a few days' illness, last summer he died and was buried in the churchyard at St. James' the Less, Falls of Schuylkill.

Dr. Vinton was born in this city, August 17, 1845. He was the youngest of the five children of Charles Adams Vinton and his wife, Elizabeth McKinley, of Philadelphia. His early life was spent in their home at 704 Sansom Street. The Vintons were of New England ancestry, and Dr. Charles was very proud of his descent from the Pilgrims. His was the eighth generation from those who sailed in the *Mayflower*, whose doughty captain, John Alden, was the first of his line from England; and his was the first generation of the family born out of New England. With such an ancestry it was natural that he should have become interested in the history of their times, and he was prone to count himself native-born. He allied himself with various patriotic and historical societies and included in his studies the antiquities of the earliest days in colonies other than that of Massachusetts.

For a number of years he and Mrs. Vinton visited in Virginia and passed the winters in the ancient house now occupied by Dr. Hunter McGuire in the famous town of Winchester. From Dr. McGuire I learned that Dr. Vinton was instrumental in having had returned to its proper place the name-plate from the casket of

Lord Fairfax, which had been lost during the transfer of the body of this last great Proprietor from building to building since its first interment. This plate Dr. Vinton found among the treasures of the Pennsylvania Historical Society, in Philadelphia, which Society graciously sent it to the Vestry of Christ Church, in 1925, in order that it might be put upon Lord Fairfax's coffin at the time of reinterment. This incident was of importance, as it was the means of identifying the casket as that of Lord Fairfax.

This Lord Fairfax, was Thomas, the sixth of his line, born in Kent, England, in 1693. His vast estate comprising 5,000,000 acres extending from the Potomac to beyond the Shenandoah and occupied that region known as the Northern Neck, on which so many flourishing towns have since arisen. He was the only English peer who had resided in Virginia, having settled there in 1747, and so continued until his death in 1781. He lived very simply and was accorded all the privileges of a Virginian citizen by the colonists, toward whose desire for independence he adopted a decidedly liberal attitude. "Mount Vernon" was adjacent; the Proprietary was the early patron of Washington, whose appreciation of the great man in later times was filled with respect and deep feeling. Fairfax was buried in Frederick Parish Church, but the body was transferred to Christ Church, Winchester, in 1828. The remains were again disturbed during the rebuilding of Christ Church but were reinterred in 1925.

Dr. Vinton was a cultured gentleman of the old school, to which he clung unvaringly in speech, manner and habit. With these attributes went along a ready keen wit and a kindly and affectionate nature which endeared him to all who knew him. Although he was a devout and learned churchman, punctilious in every observance of the services, he was tolerant of those whose ritualism was not so extreme as was his.

For many years he was a devoted member of St. Mark's Church, Philadelphia, and it was here that I became acquainted with him in my early days in the medical school. He was an active and forceful member of a *coterie*, the purpose of which was the practice of secret benevolences, and included, among others, Douglass Hall, John H. Packard, Charles Burnett and Charles Cadwalader. The meetings were held every few weeks in the Library of St. Mark's Parish House, and I was the secretary.

Dr. Vinton always prized his election to the College, and delighted

to recall the associations with the Fellows of his days, and, despite his retirement from all active practice, he unfailingly maintained his Fellowship, and he learned of the scientific interest by patient perusal of the *Transactions* year by year. He was, however, quite unknown to the present generation of the Fellows. The bequest provides that "the sum of Ten Thousand Dollars shall be invested and the income from Ten Thousand Dollars shall be applied to the purchase of books and to other purposes of the Library," while the income from the remainder "shall be applied to the uses of the Section on Ophthalmology."

It is right that the Fellows of the College should be apprised of this bequest, and that we should pay tribute to the memory of our benefactor, one who, remembering the days when he was active in the proceedings of the College, forty years ago, saw fit to make so substantial a return for the benefits he derived from his Fellowship.

MEMOIR OF JOHN B. DEAVER, M.D.*

By DAMON B. PFEIFFER, M.D.

Dr. John Blair Deaver's father was a physician. Two of his three brothers studied medicine. He was educated in the strongly clinical atmosphere of Philadelphia. Possessed of enormous vitality, finding his chief joy in intense activity of body and mind, ambitious, forceful, combining his ancestral qualities of Irish humanism with Scotch tenacity, his career was what might have been predicted from such a combination.

He was born, July 25, 1855, near Buck, Lancaster County, Penna., a strategic crossroads from which his father practised medicine through the countryside. His early education was received at the Nottingham Academy near his childhood home. He was graduated in medicine from the University of Pennsylvania, in 1878, at the age of twenty-three years. He served as intern in the Germantown Hospital and for an additional year in the Children's Hospital. He then began active practice in 1880, with an office at Sixteenth and Vine Streets. Shortly afterward he was appointed assistant demonstrator of anatomy in the University of Pennsylvania, which title he held for three years; the next two years (1883–1884) he was demonstrator of osteology and syndesmology; then for three years (1884–1887) demonstrator of anatomy; then for three years (1889–1891) demonstrator of anatomy and lecturer on topographical anatomy, and finally (1891–1899) assistant professor of applied anatomy, with which position his earlier connection with the teaching staff of the University terminated.

At that period anatomy was the recognized stepping-stone into surgery, and Dr. Deaver made full use of his opportunities to become a profound surgical anatomist and expert technician. He greatly admired Agnew, whose surgical skill was celebrated, and he never lost his interest in the technical side of surgery. Few, indeed, have been Deaver's equal, and it is doubtful if any have been his superior in the skilful use of what he was fond of calling the "aseptic scalpel."

* Read January 11, 1933.

During this early period he made many connections with Philadelphia hospitals, but his most important appointment came in 1886, when, at the age of thirty-one years, he was made surgeon to the German Hospital, now renamed the Lankenau after its greatest benefactor. Here Dr. Deaver found scope for his great energies. He rapidly built up a large clinic and entered upon the work of popularizing the new-found resources of surgery with all the enthusiasm of the evangelist. If from his many activities one might single out the most significant and important it would probably be the influence which he exerted in the spread of general knowledge and use of surgical therapy. He was successful as a teacher and quiz master, and, later, when his connection with the faculty of the University of Pennsylvania was interrupted for a period of twelve years, his bent for teaching found expression in his crowded clinics in the round marble amphitheatre at the German Hospital and in the addresses, papers and books which poured forth in ceaseless stream under his name. He was especially stimulating to young men. He had the rare inspirational quality, a gift for epigram and a virile wit. With his skill in operative technique, he transformed his clinics into a species of medical entertainment into which he interjected sallies and reminiscences often freighted with clinical wisdom that remained indefinitely in the minds of his hearers. He liked young men and they looked up to him as a demigod. He came to exercise a somewhat similar influence upon the rank and file of the profession. No one understood their psychology better than he, and he was thus greatly instrumental in spreading that belief in surgery which was his gospel. Especially was this true in connection with acute abdominal conditions, notably appendicitis, with which his name became so inseparably associated as one of the pioneers. Many owe their lives to his skilful surgery, but many more to his powerful advocacy of the early operation.

In 1911 he was recalled to the University as professor of the practice of surgery, and, in 1918, became John Rhea Barton professor of surgery, holding this position for four years, the retirement age having been extended for him in recognition of his vigor and value to the University. On one occasion Moynihan referred to the ancient ideal of a surgeon as one who possesses the "heart of a lion, the eye of an eagle and the hand of a woman," to which Deaver at once added "and the constitution of a mule." This was one of his greatest assets. From his Scotch-Irish forebears he

had inherited a physical endowment that enabled him to go at top speed through life, with full realization of the joy of living. He rarely knew fatigue and never acknowledged it. So intensely alive was he that it is difficult for those who knew him to believe him dead. He passed away, September 25, 1931, as a result of a rapidly progressive anemia, the cause of which was never fully known, but probably had a background of obscure infection.

Dr. Deaver was a member of many societies. To trace his various activities would be a long task. He never sought honors, but was happiest in doing, talking or thinking surgery. For the most part he shunned official positions, having no taste for directing or organizing mass activities. Still he could not avoid all recognition of this sort. He received a number of honorary degrees and was president of the American College of Surgeons and the Post-Graduate Medical Association of North America, honorary positions which did not drag him away from the wards and his beloved clinic. Beside his books on anatomy, appendicitis, the breast, the prostate and the surgery of the upper abdomen, he contributed to medical journals a total of 248 articles on a wide variety of subjects. He was in great demand for clinics, for discussions and addresses, to gatherings of physicians, in the course of which he traveled extensively. This sort of outing he enjoyed. Vacations in general he loathed and, if necessary, he resorted to stratagem to avoid or shorten those periods of relaxation which those who were anxious for his welfare occasionally induced him to take. Like the great, he was simple in his tastes, and, with Cicero, he considered nothing human foreign to him. He did what he liked because he liked what he did. To surgery he gave a full measure of devotion. To others he was always on call in their hour of need.

Surely he must be accorded a high place in the Valhalla of Philadelphia medicine.

MEMOIR OF CHARLES KARSNER MILLS, M.D.*

By THEODORE H. WEISENBURG, M.D.

It is given to few scientific men to make an impression on their particular specialty, for most of us make no impress whatever, but Charles Karsner Mills definitely led neurological thought in this country from 1890 to 1920, a period of about thirty years. While the character of scientific investigation does not, as a rule, depend upon periods, nevertheless Dr. Mills was fortunate to be born in the golden age of cerebral localization. Stimulated by the writings of Hughlings Jackson, the greatest neurological philosopher of all time, his work and accomplishments were chiefly in that field, and he has been well called the Hughlings Jackson of America.

His publications show the wide range of his interests, his neurological contributions alone numbering 345. This does not take into consideration a textbook of *The Nervous System and Its Diseases*, perhaps the most philosophical and scholarly volume on the subject that has appeared in the American literature. He also wrote two other books of less interest, one of which, *A Textbook on Physiology and Hygiene*, a product of his early life, was widely used and translated into many languages.

Dr. Mills' influence on neurological thought in this country was not only evidenced by his many contributions, but by his interest in many organizations, particularly the American Neurological Association, which honored him by electing him twice president, the second occasion on its semi-centennial anniversary which to do him honor was celebrated in Philadelphia. His love for this Association was such that in his membership, covering a period of fifty years, he missed only one meeting. The American Neurological Association, in recognition of his outstanding contributions to American neurology has decided to honor his memory by the creation of a Charles Karsner Mills memorial lectureship.

His influence on Philadelphia neurology was profound. He founded the nervous wards in the Philadelphia General Hospital,

* Read January 11, 1933.

which now house about 500 men and women patients, perhaps the best collection of neurological material in the world. Much of Dr. Mills' work as well as that of most neurologists in Philadelphia is based upon material from these wards. The influence that these wards have had upon Philadelphia neurology can hardly be overestimated. In 1883 he founded the Philadelphia Neurological Society.

His teaching career began in 1877, when he was appointed lecturer on electrotherapeutics in the medical department of the University of Pennsylvania. From that time on until his death he was continuously connected with the University in an active teaching capacity, but his happiest period was from 1901 to 1915, when he was head of the Neurological Department. He surrounded himself with the most active and promising men in Philadelphia, and so meritorious and numerous were the contributions from his department that the Philadelphia school of neurology was recognized all over the scientific world.

Dr. Mills had a very active practice and was an eminent consultant. As a medico-legal expert his services were eagerly sought for. He was a witness in many celebrated cases. His interests, however, were by no means limited to neurology, for he was one of the most scholarly and cultured physicians in Philadelphia, contributing extensively to medical biography and history.

The great interest of his life, however, was the advancement of scientific neurology. He sacrificed everything for it. On numerous occasions during his office hours he would become interested in a discussion and dismiss an office full of patients. His enthusiasm was such that any young physician with neurological ambitions naturally came to him, for he never failed to help, encourage and advise, and he has the distinction of developing more good neurologists than any man in his time.

Dr. Mills was proud of his military career. In 1862, at the age of seventeen years, while still a student in the Philadelphia Central High School, he served in the Emergency Campaign with the 8th Civil War Regiment, and in 1863 with the 33d Regiment. Dr. Mills had the unique distinction of writing his own discharge when his services were terminated as a corporal, for he also acted as clerk of the Company.

Despite his busy life Dr. Mills, in 1864, began to keep a scrapbook. He kept this up, accumulating thirteen volumes, which

recorded not only his progress, but that of contemporary times. The last addition which he made was a note of the death of his life-long friend, Dr. Francis X. Dercum, which occurred about a month before his own demise. His daughter completed the record with the account of his own death and funeral.

Dr. Mills also wrote an autobiography. This occupied the closing years of his life and was almost completed. He worked upon it a few days before his death.

Dr. Mills' success was all the more remarkable when it is considered that from his earliest childhood he was handicapped by poor vision, and that for about half of his lifetime he was unable to read. Perhaps his many friends will remember best his personal charm and delightful sense of humor. He belonged to the few who are able to laugh at themselves. He was always courteous and considerate of the feelings of others. He had great courage and fought for his convictions.

On May 28, 1931, in his eighty-sixth year, Dr. Mills died from the results of the effort he made to attend the annual meeting of the American Neurological Association held in Boston. He died fighting for the great passion of his life—neurology.

THE PRESIDENT'S ADDRESS*

By FRANCIS R. PACKARD, M.D.

As there is much absolutely necessary business to be transacted by the College this evening, I shall make the customary address of the President as short as possible.

On December 1, 1931, our Fellowship consisted of 526 Fellows, of whom 483 were Active, including 43 Nonresidents and 19 Associate Fellows. Since that date we have lost by death 16 Active Fellows, including 2 Nonresident Fellows and 2 Associate Fellows, a sadly heavy mortality, and by resignation 6 Active Fellows. In spite of this on December 1, 1932, our Active Fellows numbered 496, an increase of 13, of whom 42 were Nonresident Fellows, and there are 17 Associate Fellows. I believe that the College should have a much larger number of Active Fellows. During the past year a by-law was adopted extending the limit of the distance from Philadelphia within which Active Fellows must reside, from thirty to seventy-five miles and abolishing the requirement that they should be residents of the State of Pennsylvania. This liberal step it is hoped will bring into our Fellowship some of the many eligible men who live in New Jersey or within the more extended radius from Philadelphia.

I think it is the duty of every Fellow of the College to seek out eligible candidates for election, especially among the younger members of the profession, and propose them for Fellowship. I also cannot impress too strongly on those who propose candidates for election that if they wish to secure the election of their candidate they should attend the business meeting of the College at which his name comes up, in order that they may vote for him and further his interests among the other Fellows. At the semi-annual business meeting of the College on May 9, 1932, five candidates who had been recommended for election by the Council failed to receive the necessary four-fifths vote. One or two votes would have turned the balance in favor of each of these candidates. But so few of the

* Read January 9, 1933.

Fellows who had proposed the candidates in question attended the meeting, that as a result of their absence the men they were presumed to be interested in were rejected by the College.

During the year the College has received the legacy of $5,000 left by the late Dr. Hobart A. Hare. Three of our Fellows, Drs. W. W. Keen, A. P. C. Ashhurst and Charles M. Montgomery, who died during the year, have left bequests to the College.

During the past year the College has received many valuable gifts, including a volume of the First TRANSACTIONS of the College from Dr. B. A. Robinson, of South Amboy, N. J.; a number of manuscript notebooks and letters written by Dr. Hugh Lenox Hodge, the gift of Dr. Edward B. Hodge; some manuscripts of Dr. Joseph Carson from Mrs. Hampton Carson; a letter written in 1884 by Dr. Samuel W. Gross to Dr. Benjamin Parish, given by the latter's son, Dr. Benjamin Parish; a cane, seal, and other articles which belonged to Dr. Nathaniel Chapman, from Mrs. William Philler and Miss Mary Winsor, and a case of postmortem instruments which belonged to and were used by Dr. Horatio C. Wood, and were given by him to Dr. William G. Spiller, presented by the latter.

Dr. Jay F. Schamberg presented to the College nine volumes containing the typewritten stenographic reports of the Pennsylvania State Vaccination Committee which was appointed by Governor Tener in 1912. There were but seven copies of these reports and as they contain the testimony of hundreds of the most prominent physicians and scientists they are of great historical importance.

MEETINGS. Three business and 8 scientific meetings were held during the past year. On January 4 a meeting was held under the auspices of the James M. Anders Fund, at which Dr. Simon Flexner, of New York, spoke on "The Poliomyelitis Epidemic of 1931." On November 2, the eleventh Nathan Lewis Hatfield Lecture was delivered by Dr. R. Tait McKenzie on "The Facial Expression of the Emotions, with Special Reference to Violent Effort and Fatigue," on which occasion Dr. McKenzie generously used the honorarium from the Fund to have four of his well-known works cast in bronze and gave them to the College. The regular meeting on December 7 was devoted to the Mutter Lecture, which was delivered by Dr. H. Gideon Wells, of Chicago, "Summarization of the Advance in Our Knowledge of Some Features of Cancer." Several meetings were

assigned to interesting symposia on special subjects: That on February 3, under the auspices of the Section on Public Health and Industrial Medicine, dealt with various problems connected with health service to the community and medical economics. On April 6 there was an interesting symposium on various aspects of the tuberculosis problem, and on May 4 one on cancer. The recent outbreak of poliomyelitis in Philadelphia was the subject of a symposium on October 5. The average attendance of Fellows at the meetings was fifty, an increase of six over the average of the previous year.

THE LIBRARY. Our library now contains 169,145 bound volumes, an increase of 2339 volumes during the year. The incunabula in the library now number 406, and our collection of portraits has increased to the immense number of 23,441. This portrait collection, including pencil and pen-and-ink sketches, silhouettes, engravings and photographs, is of the greatest value. It is continually being utilized by authors and lecturers who wish to reproduce pictures. The library has been open as heretofore two evenings each week and on the minor legal holidays.

In June Mr. Charles Perry Fisher resigned the position of librarian and superintendent of the hall of the College of Physicians after fifty years of unwearied devotion to its interests. In September, 1882, Mr. Fisher was appointed assistant to the librarian of the College. At that time the post of librarian was held by Dr. Frank Woodbury. In the following year the position of honorary librarian was created and Dr. James P. Hutchinson elected to fill it, and Mr. Fisher was appointed librarian. During the half century which ensued the library increased from 25,000 to over 160,000 volumes, but it is not only because of the increase in numbers of volumes that we are proud of it; it is the quality, the character of our collection, which marks its especial value and much of this is due to Mr. Fisher's knowledge and skill. We not only possess one of the best collections of medical incunabula, but our collection also contains most of the important editions of epoch-marking medical works. Thus our collection of editions of Harvey's writings excels even that in the Royal College of Physicians of London, and Mr. Fisher's bibliographic list of its items is referred to repeatedly by Keynes in his Harvey *Bibliography*, and we have also very complete sets of the various editions of Vesalius, Sydenham and other authors which belong to Osler's *Bibliotheca Prima*. To Mr. Fisher we owe the

establishment of the card-index catalogue, and it was primarily due to him that the library was among the first libraries to install a photostat. Mr. Fisher was one of the founders, in 1898, of the Association of Medical Librarians, which subsequently became the Medical Library Association, of which he once was president, and in whose proceedings he has always borne an active part. He is universally recognized as one of the foremost medical librarians of the world.

After Mr. Fisher's resignation Dr. Emlen Wood, who had previously held the position of assistant to the librarian, was appointed acting librarian, and Miss Mary E. Zelner was appointed superintendent of the hall.

The Mutter Museum. The museum continues to be increasingly used by teachers and students. Its collections have been increased and their use broadened in many ways, owing to the zeal of the curator, Dr. Harry P. Schenck, and the interest of the committee in its affairs.

The Alvarenga Prize. The prize was not awarded this year, as none of the essays submitted in competition were deemed by the committee to have met its requirements.

The Nurses Directory. On the afternoon of Saturday, May 14, an interesting meeting was held to celebrate the fiftieth anniversary of the founding of the Nurses Directory of the College. A large audience attended, and interesting addresses were delivered by Drs. William J. Taylor and Frederick Fraley.

During the half century in which it has been in operation the Directory has not only been of great use to the profession and to the public, but has been a great factor in maintaining the standards of nursing in this city. The committee has exercised the greatest care in accepting applicants for registration and in its oversight of those who are registered. Any complaint about the conduct or ability of a nurse is given careful attention and the impartiality of the members of the committee has placed it in a position to act with justice to them and to the College. During the past fifty years the College has received from it the large sum of $97,616.34. Of course, it has felt the prevailing economic depression, but even during the past year it has had on its registry in good standing 536 women nurses, 48 certificated nurses, 48 male nurses and 157 attendants, a total of 789, plus 6 physiotherapists, and this in spite of the economic depression and of the rivalry with the newly established directory

under the control of the nurses themselves and of the numerous directories in the various hospitals.

THE SECTIONS OF THE COLLEGE. These have been, if anything, more active than usual and have continued to maintain the high standard of their meetings. In some instances they have broadened their scope with great advantage. Thus on November 16 the Section on Oto-Laryngology held a joint meeting, in Thompson Hall, with the Section on Laryngology of the New York Academy of Medicine as its guests and joint participants at which there was a very large and enthusiastic attendance. The Section on Medical History held three meetings in Mitchell Hall, one in celebration of the bicentennial of George Washington, a notable meeting on the history of the development of physical diagnosis and a third dealing with the history of the Philadelphia Hospital.

I still find a deplorable lack of coöperation between the officers of the Sections and the Committee on Scientific Business of the College. I believe that both the interests of the Sections and the College would be greatly helped if they kept in closer touch with one another and made some effort to organize their meetings on a more definite plan.

It is the sad duty of your President at this meeting to say a few words about some of the Fellows of the College who have departed from us. I am sorry to say the list this year is a long one.

Dr. G. Victor Janvier was elected a Fellow of the College December 1, 1920. After graduating in medicine from the University of Pennsylvania, in 1906, Dr. Janvier practised in Lansdowne, Delaware County, Pennsylvania, devoting himself especially to obstetrics and pediatrics. He was also much interested in public health movements, serving as physician to the Board of Health of Lansdowne, and being very active on the Board of Associated Charities of Delaware County. During the last years of his life he became much interested in the subject of birth control and conducted a clinic devoted to it. Dr. Janvier died after a short illness on December 17, 1931.

Dr. Richard Sydwell Hooker, who was elected a Fellow of the College on January 7, 1914, died on December 25, 1931, aged fifty-four years. Dr. Hooker was a quiet, unassuming man, of scholarly tastes. He served as resident physician at the Orthopedic and Episcopal hospitals, and was later neurologist to St. Christopher's Hospital and the Roosevelt Hospital and physician to the out-

patient department of the Episcopal Hospital. He contributed several papers on neurology to the literature of the subject. At his death a very valuable collection of books and pictures bearing chiefly, not on neurology, but on dermatology, which he had gathered and carefully classified, was presented by his widow to our College.

Dr. Thomas C. Ely was elected a Fellow of the College on June 3, 1896. After graduating from Colgate University, Dr. Ely received his medical degree from the University of Pennsylvania in 1887, and practised in Philadelphia until his death, at the age of sixty-eight, on December 26, 1931, from an overdose of a sedative, accidentally taken. Dr. Ely had a large practice and was greatly beloved by his patients and friends.

Dr. George C. Stout, elected a Fellow of the College on December, 1898, died on January 27, 1932. He was born in Bethlehem, Pennsylvania, on June 29, 1862. He graduated from the U. S. Naval Academy at Annapolis in 1883, but after some years in the Navy he took up the study of medicine and graduated from the medical department of the University of Pennsylvania in 1891. After a course of postgraduate study in Europe, Dr. Stout settled in Philadelphia, where he devoted himself exclusively to the practice of laryngology and otology, in which he achieved great success. For many years Dr. Stout was chief of the department of otology and laryngology of the Presbyterian Hospital. He was an active Fellow of the American Laryngological Association, and a former chairman of the section on laryngology and otology of this College. Owing to ill health, he had retired from active practice some years before his death which was due to heart disease.

Dr. Francis B. Jacobs, elected a Fellow of this College October 3, 1906, fell dead from his horse while riding in a fox hunt near West Chester, Pennsylvania, on February 11, 1932. Dr. Jacobs had lived all his life in West Chester, where he practised pediatrics. After graduating from Haverford College in 1897, and from the medical school of the University of Pennsylvania in 1900, Dr. Jacobs served as a resident physician in the Pennsylvania Hospital. Although living at a distance from the city he was an active participant in meetings of the medical societies in Philadelphia. Dr. Jacobs was fifty-eight years of age at the time of his death.

Dr. Charles M. Montgomery was elected a Fellow of the College April 1, 1908. After graduating from the college department of the

University of Pennsylvania in 1897, he received his medical degree from the same institution in 1901, and then served as resident physician in the Pennsylvania Hospital. He received a commission as captain in the Medical Reserve Corps of the United States Army in April, 1917. In spite of the handicap of ill health, Dr. Montgomery remained on active service until September 30, 1920, when he was honorably discharged. After many years' illness from pulmonary tuberculosis, he died at Oteen, N. C., on March 3, 1932. Dr. Montgomery's affection for our College led to his bequeathing us a sum of money in his will.

Dr. Thomas Appleton Cope was elected a Fellow of the College on January 11, 1932. Dr. Cope graduated from the Medical Department of the University of Pennsylvania in 1900. For some years he was an instructor in medicine at the University and fellow in bacteriology in the William Pepper Laboratory of Clinical Medicine. Later he was an associate professor of medicine in the University and assistant professor of medicine in the Graduate School. He also was lecturer on clinical pathology in the Woman's Medical College and pathologist to the Germantown Hospital. Dr. Cope was in the fifty-fourth year of his age when he died suddenly of cardiac disease on May 27, 1932.

Dr. Orlando H. Petty, who was elected a Fellow of the College on October 2, 1913, was born in Cadiz, Ohio, in 1874. After graduating from Jefferson Medical College in 1904, Dr. Petty served as resident physician in St. Timothy's Hospital, and then practised for about a year in Johnstown, Pennsylvania. Returning to Philadelphia, he devoted himself to private practice and work in disorders of metabolism, and won special recognition by his success in the treatment of diabetes. In 1924 he published a book on diabetes, which went through five editions. In 1917 he was appointed a surgeon in the Marine Corps with the rank of captain, and went overseas with the fifth Marines, second Division, in August, 1917. Dr. Petty was one of the few physicians who have ever been awarded the Congressional Medal of Honor. It was awarded to him for his bravery on June 11, 1918, when he worked at a front-line station under heavy shellfire and carried a wounded officer off the field under heavy gas exposure. His health was permanently and severely damaged by the gas from which he suffered on this occasion. Dr. Petty was also awarded the Distinguished Service Cross, the Croix de Guerre with palms, and the Italian War Cross. Dr. Petty, at

various times, taught at Jefferson Medical College, was professor of diseases of metabolism in the Post-graduate School of the University of Pennsylvania, and in charge of the department of metabolism of the Philadelphia General Hospital. For a short time he was Director of Public Health of this city. He was a thirty-second-degree Mason, and at one time president of the County Medical Society of Philadelphia, and of the Medical Club of Philadelphia. Exhausted mentally and physically by many years of dire illness, aggravated by persistent hard work, Dr. Petty took his life on June 2, 1932.

Dr. Harold M. Whiteway was elected a Fellow on February 1, 1905. An Englishman by birth, Dr. Whiteway came to America in his youth. After graduating in medicine from the University of Pennsylvania in 1899, he served as resident physician at the Polyclinic Hospital, and then practised medicine in Philadelphia for some years, after which he removed to Los Angeles, California, where he died on June 6, 1932. A gentleman and a cultured scholar, Dr. Whiteway was much missed by a large circle of friends and patients after his departure from Philadelphia.

Dr. Joseph Leidy, who was elected a Fellow of the College, November 2, 1892, died of heart disease at his home in Penllyn on July 6, 1932. He was born on April 11, 1867. He was a nephew of the famous naturalist, Joseph Leidy, and was generally known as Joseph Leidy, Jr. After graduating from the University of Pennsylvania in the medical class of 1887, Dr. Leidy served as resident physician first in the University Hospital and afterward in the Pennsylvania Hospital. He was at one time on the staff of the Howard Hospital, and for some years an assistant demonstrator in pathology and morbid anatomy in the University Hospital. During the World War he entered the army medical service as a captain, and later was promoted to major. For many years before his death Dr. Leidy was engaged in writing a life of Leidy, the naturalist. Fortunately he left the material he had accumulated in such shape that the work can be readily brought to publication.

Dr. Max Henley Bochroch was elected a Fellow of the College on March 7, 1894. Dr. Bochroch was born in Wilmington, Delaware, on March 7, 1861. He graduated from Jefferson Medical College in 1880, and settled in practice in Philadelphia. Devoting himself entirely to neurology Dr. Bochroch contributed many articles on that subject to current medical literature and served as an expert

witness in many legal cases. He was a member of the staff of the Philadelphia General Hospital, the Jewish Hospital, and St. Joseph's Hospital, and professor of psychiatry in Temple University School of Medicine. He died at his home in Philadelphia on November 27, 1932, of heart disease.

Associate Fellows. Dr. Frank Billings, of Chicago, who died September 30, 1932, was elected an Associate Fellow of the College November 3, 1909. Dr. Billings was one of the most prominent physicians of our time, distinguished as a research worker, clinician, teacher and administrator. He was one of the first men in this country to realize the importance of Koch's discovery of the tubercle bacillus and to demonstrate the technique of the examination of the sputum for finding it. He served as professor of medicine at different times for many years in the Northwestern University Medical School and in Rush Medical College. He was very active in the affairs of the American Medical Association and served two terms as its president. To Dr. Billings is owing a large share in elevating the Association to its present status. He was president of the Association of American Physicians. It would be impossible to enumerate the many other positions with which his associates delighted to to honor him in his active career. Dr. Billings made many most important contributions to medical literature, and was active in organizing many institutions and societies which have had an important bearing on medical science. During the World War he took an active part as consultant and advisor to the Government, and in 1917 went to Russia as chairman of a Red Cross commission to study the food and sanitary conditions of that country. He was seventy-eight years of age when his death occurred from gastric hemorrhage.

Sir Ronald Ross, who died on September 16, 1932, was elected an Associated Fellow of the College, February 5, 1908. The son of an officer in the British army, Ross was born in India in 1857, and after getting his medical education in England he passed a large part of his own life in the medical service of the British army in India. A man of acute intellect and much cultivation, he occupied his leisure hours from his professional duties in writing a number of plays and some poems of great merit and he was also a student of and writer on the higher mathematics. Becoming deeply interested in the rôle played by the mosquito in the transmission of malaria, in 1897–1898 he succeeded in unveiling the life history of the para-

site. Ross retired with rank of major. In subsequent years he conducted many expeditions to tropical countries to study problems connected with their diseases. He taught in the Liverpool School of Tropical Medicine and in King's College, London. During the World War he went to Egypt and to the Dardanelles as a chief consultant to the army. In 1902 he was awarded the Nobel Prize. The Hospital and Ross Institution for Tropical Diseases at Putney was founded through his efforts and named in his honor. Although he was knighted and received many other honors, the last years of Ross' life were passed in much distress owing to illness and straightened resources.

POLIOMYELITIS*

Mode of Infection and Means of Prevention

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I was happy to accept the kind invitation to deliver the James M. Anders Lecture and to speak on the subject of poliomyelitis. The inherent interest in the disease has been increased by the severe epidemic which prevailed in New York and other eastern states during the past summer and autumn period. The epidemic of 1931 in New York State as a whole was only less severe than the unprecedented outbreak of 1916, when in New York City alone 9345 cases were reported. It is now quite generally recognized that this number, large as it is, is smaller, perhaps much smaller, than the number of cases which actually occurred. Inasmuch as many cases manifest themselves in the form of minor illnesses only, the reporting made to the health authorities is always incomplete. In the same way the reported fatalities arising from the epidemic disease, when calculated in percentages, have approximate value only. Obviously, the larger the number of mild cases reported, the smaller will be the relative mortality. If a comparison is made of the mortality of the 1916 epidemic in New York City, which was placed at 26.24 per cent, with that of 1931, given at 12.15 per cent, the conclusion might be drawn that the later outbreak was milder than the earlier one, which is by no means necessarily the case. It is highly probable that many more mild cases were recognized by physicians in the epidemic of 1931 than in that of 1916.

It is well to review our knowledge of epidemic poliomyelitis at this time. In so doing it is proposed to deal with the subject under two general headings: the mode of infection and the means of prevention. In this way we are in a position to consider present knowledge of the disease in the light of the public health measures

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employed for its control. Poliomyelitis has prevailed periodically in the United States since 1907–1908, when the disease was brought into this country from Europe. The immediate predecessor of the general European and the United States outbreaks was the epidemic of Norway and Sweden in 1905. That particular epidemic, while of alarming severity, was not unheralded. Ever since 1868 the disease was known to prevail endemically in those two countries, gaining in severity decade by decade. The outbreak in Stockholm in the earlier period led to the publication of Medin's[1] article in which the infectious nature of the disease is stressed, just as the epidemic of 1905 led his pupil Wickman, in that monograph[2] which has now become a classic, to redefine and classify poliomyelitis, and to emphasize the occurrence and significance of mild or abortive cases for the spread of the infection.

It may, therefore, be said that a quarter of a century has passed since poliomyelitis first displayed pandemic potentialities and came to be studied by modern bacteriological methods. This period has seen a rapid growth in knowledge of the pathology of the disease, using the term in a wide sense. The clinical character of poliomyelitis has come to be not only more accurately and broadly defined, but the medical profession has learned a vast deal in regard to a more complete and certain diagnosis. Perhaps the greatest advances have been made in respect to the etiology, and for want of a better, simpler term, in what may be called the epidemiology of the disease. These advances depended in the first instance on successful communication to a lower animal, accomplished in 1908 by Landsteiner and Popper,[3] and by Flexner and Lewis.[4] Landsteiner made the first successful inoculation of poliomyelitis into the monkey; and Flexner and Lewis first accomplished serial monkey to monkey passage by means of the intracerebral method of injecting infectious nervous tissues. It may be of interest to mention that the two specimens of human spinal cord designated as "M.A." and "K.," employed originally by Flexner and Lewis, are still represented in the virus inoculum used for experimental investigations.

Poliomyelitis is a virus disease.[5] The filterable, invisible virus is present in all parts of the central nervous system of human beings and monkeys suffering from the malady, and it is present also, but more difficult to detect, in the nasopharyngeal mucosæ.[6] The detection of the virus is accomplished only through its inoculation into monkeys; aside from its power to produce paralytic

poliomyelitis in these animals, which accurately reproduces the corresponding disease in man, there are no means known of demonstrating the existence of the virus.

Mode of Infection. The manner in which infection occurs has been studied primarily in the monkey, and the results obtained in this way have been tested, as far as is possible, by the study of materials secured from human cases of poliomyelitis during life and at autopsy after death. Wickman suggested two portals of entry of the, at that time undefined, infectious agent into the body: the respiratory and the gastrointestinal tracts. These portals are still being discussed as avenues of entrance of the virus. Other external sources of infection were brought under consideration later: carriage of the virus by animals living in near association with man and insect vectors. In order to save time, it may be stated that no competent evidence has been adduced in support of the insect vector conveyance or animal reservoir theory of the virus. The more accurately these sources have been investigated, the less have they withstood critical examination. The exceptionally wide distribution of poliomyelitis also throws doubt on these means of infection. It is now known that while poliomyelitis is mainly a disease of temperate climates, it prevails epidemically in tropical islands and even in cold regions, as, for example, Iceland.[7]

The question of animal carriers of the virus has received considerable attention. Flexner, and Amoss and Haselbauer[8] early examined the common domestic animals, without securing evidence of the presence of the virus in the nervous system. They gave special attention to vermin, that is, rats and mice, obtained from localities in New York in which many cases of poliomyelitis were occurring; and they studied the power of the virus to survive when injected into the brain of several animal species. These studies gave no support to the animal-reservoir theory.

A more rigorous examination of this kind was carried out during the epidemic of 1931. Wild rats and mice were trapped in regions in which cases of poliomyelitis were occurring daily. It is of some interest to record that in the older, more closely built up portions of Brooklyn, which was one of the most active foci of the epidemic, it proved impossible to trap these animals; but they were caught in numbers in the more outlying, newer, and less densely populated districts in which cases of the disease were also arising daily, although in fewer numbers. The rats and mice were employed in two ways

in an effort to secure evidence of the presence of the virus. In one investigation the brain, spinal cord, liver, spleen, kidneys and lymph glands of the animals were inoculated into Macacus rhesus monkeys. In another investigation, poliomyelitis virus was injected into the body of rats and mice, in order to determine its capacity to survive in these animals. For the purpose of completeness, two kinds of virus were used for the inoculations: the adapted monkey passage strain, which is highly infectious, and human spinal cord recently obtained from fatal cases of poliomyelitis. As the virus undergoes modifications in its passage through monkeys, this precaution seemed necessary, since the power to survive might possibly differ in the two strains.[9]

Before presenting the results of these tests which were more rigorously carried out than the earlier ones, it is desirable to describe briefly a phenomenon which makes the detection of the virus more delicate and certain. In general, it may be stated that the virus, as it exists in human nervous tissues, is implanted in the monkey with difficulty. In any given epidemic, a part only of the specimens of spinal cord from acute, fatal cases can be so implanted. Even when inoculated successfully, the further or serial transmission of the virus varies. For reasons not understood, certain specimens can be readily passed, and in the course of passage become highly active or potent; others quickly lose the power of inducing infection. However, by resorting to a device which we have called "acceleration,"[10] a weak virus, or a strong virus given in a dose below that of the minimal infective one, may now often be detected. The device consists in reinjecting inoculated monkeys with the same specimens at the expiration of from seven to ten days, which interval represents the average incubation period. Following the accelerating dose, fever (up to 104° to 106° F.) appears in from one to three days, and symptoms of poliomyelitis arise. We have used this method of establishing refractory human strains of the virus in monkeys and in detecting weak passage strains and the presence of quantities of the strong virus below the minimal effective dose.

The acceleration device, as outlined above, was employed in the examinations referred to of rats and mice, as well as young rabbits, but with negative results, with the exception of one instance, in which a potent virus was found to survive in a rat up to the tenth day. We feel justified, therefore, in concluding that these rodents do not at certain periods and under certain circumstances serve as

animal reservoirs for the dissemination of the virus of poliomyelitis in the environment of man.

There are left for examination, according to the views variously held regarding the sources from which the virus enters the body on its way to the central nervous system, two portals of entry: the upper respiratory system and the gastrointestinal tract, which will now be considered.

Gastrointestinal Infection. It will be recalled that Wickman was of the opinion that the infectious agent enters the body through both the digestive and the respiratory tract. The first advance in knowledge came with the discovery that the virus, sealed in the brain of monkeys, penetrates into the nasopharyngeal mucous membranes.[6] This observation was followed by two sets of confirmatory tests: (*a*) The infection of monkeys by way of the nasal mucosa through intramembrane inoculation or mere instillation of the virus,[11] and (*b*) by the demonstration of the virus in washings from the nasopharynx and in the excised tonsils of man.[12] In the latter instance washings from healthy contacts were shown to contain the virus, indicating that passive human carriers exist and doubtless play a part in its distribution.

On the other hand, the early efforts made to induce experimental infection by feeding the virus to monkeys were fruitless.[13] A single exception to the rule is to be noted. Leiner and von Wiesner[14] reported two successful tests. Monkeys (the species not being stated) fasted for from twelve to twenty-four hours received an administration of opium to control peristalsis, and were then given an injection of virus into the small intestine; or they were fed virus by the stomach tube. Of 4 laparotomized monkeys, 3 became paralyzed; of 2 fed monkeys, 1 responded and the other resisted infection. The symptoms and histological lesions were characteristic of experimental poliomyelitis.

The results of these tests seem to accord with the few instances in which it has been shown that milk may serve as the vehicle for the distribution of the virus.[15] They conform also to the recently expressed view of Kling[16] that poliomyelitis may at times be a water-borne disease, and they seem to be supported by the experiments reported by Kling, Pettersson and Wernstedt in 1912,[17] in which filtered intestinal washings from man induced the experimental disease in monkeys. Moreover, the European epidemics of poliomyelitis from 1928 on led Levaditi, Kling and Lépine[18] to

carry out a larger set of feeding tests on Macacus cynomolgus, a species which they consider more favorable for the tests than the Macacus rhesus, usually employed. The feedings were made on successive days by stomach tube and directly. The excretion of viable virus in the dejecta was investigated. A relatively high proportion of positive or infection tests was reported from the feedings, and a number of positive results in the elimination tests.

Since 1910 the question of the gastrointestinal portal has been repeatedly studied at the Rockefeller Institute.[19] An effort made in 1912 to confirm the positive tests of Kling, Pettersson and Wernstedt failed wholly. The experiments were carried out even more rigorously and, indeed, favorably than those of the Swedish investigators, since not only intestinal contents, but also the mucosæ, including the enlarged lymph follicles obtained at autopsy soon after death, were employed for inoculation, after comminution and filtration and after destruction of bacteria by means of 0.5 per cent phenol which does not destroy the virus. Finally, monkeys were fed the highly potent, adapted virus, after which washings of their intestines were made, filtered and inoculated. With the exception of 1 monkey, which became paralyzed, the other tests proved negative. Contrary, therefore, to the reports of the Swedish authors, the conclusion was drawn that in man or the monkey the virus of poliomyelitis does not readily survive in and enter the body by way of the gastrointestinal tract. In 1913 the tests were repeated by us on Macacus cynomolgus, but still without success.[19]

With the outbreak of the epidemic of 1931, this important question was reëxamined, both human and monkey specimens being studied at that time. The most reliable results were obtained with the latter. The virus originally present in human spinal cord and brain has low potency for monkeys, only a few strains becoming highly virulent after serial monkey passage. Even then, close attention is required, since these active strains sometimes undergo sudden fluctuation and reduction of activity. The tests made of human specimens included the mesenteric and other lymph nodes, which proved noninfective even after the acceleration method had been resorted to. This observation is in conflict with an early experiment[20] in which virus had been detected in the mesenteric glands. In 1931 the nodes were removed at autopsy with great care to avoid contamination, and we now suspect that there had been accidental virus contamination of the earlier specimen.

Six Macaci cynomolgi were fed the highly potent passage virus by stomach tube. The feedings were repeated on six successive days, and the quantity of virus given on each occasion was many times the dose infective by intracerebral inoculation. Of these 6 animals, 1 developed paralysis of one leg which, contrary to the rule in infected monkeys, failed to progress after the first few days. This animal recovered with a residue of atrophy of the leg muscles.

Monkeys which are fed virulent material tend not to eliminate living virus. The perfection of the *in vacuo* concentration method by Clark and his associates[21] has made it possible to use large amounts of filtrates and thus to secure material favorable for inoculation. There is, however, a limit to the amount of virus that can be quickly destroyed by the stomach and intestine. Thus, if from 6 to 25 grams of virulent monkey spinal cord are fed and the feces are collected during the following forty-eight hours, extracted, and the filtered extract is concentrated *in vacuo*, an infective inoculum may thus be secured.[19]

Before passing to the subject of the respiratory portal of entrance of the virus, it is desirable to point out that our studies have not upheld the gastrointestinal tract as an avenue of conveyance of the virus to the central nervous system. It is apparent that the studies made at the Rockefeller Institute do not confirm those of the Swedish and other European investigators. They seem also not to be in accord with the well-attested milk-borne epidemics, but it is doubtful whether they are really in conflict with these. It is to be borne in mind that when milk is drunk the pharyngeal mucosa becomes contaminated with any bacteria or like organisms contained in it. This is well illustrated by happenings in diphtheria, scarlet fever and septic sore throat. In drinking, young children also regurgitate fluids into the postnasal cavities. The questions, therefore, of milk conveyance and of experimental feedings are quite different and should not be confused. Even with tube-fed monkeys, care and skill are required to avoid regurgitation and to avoid even minute injury of the mucous membranes, from which virus may be carried to adjacent nerve structures. When virus is injected directly into the small intestine after laparotomy, the difficulty of avoiding nerve contact is greatly increased. All these considerations taken together throw not a little doubt on the successful experiments reported as upholding the alimentary source of infection in poliomyelitis.

Nasopharyngeal Infection. Investigations thus far reported have led either to wholly negative conclusions or, at best, to equivocal results. We turn, therefore, to a more promising line of inquiry. The upper respiratory mucous membranes serve as the place of entrance into the body of a large variety of injurious agents bacteria, toxins, allergens and viruses. Early in the experimental study of poliomyelitis an observation was made which has been followed since with ever-increasing profit. It was found that virus injected into the brain of monkeys (after which the small trephined opening is sealed) escapes into the nasopharyngeal mucosa.[6] Disease inciting microörganisms must of necessity have a way of escape from, as well as entrance into, the body if they are to continue to be effective. By following the manner of escape, investigators may in difficult instances be led to the discovery of the manner of entrance. It was quickly found that the virus of poliomyelitis applied to the scarified nasal membrane, or merely instilled by dropper into the nares, sets up experimental poliomyelitis with only less regularity than after an intracerebral injection.[22] The disease runs identical courses in both instances; the distribution of paralyses is similar, the lower extremities becoming paralyzed more often than the upper. In general, the monkey develops a more widespread and fatal disease than is common in man, the four extremities and back muscles of the animal, and not infrequently the facial muscles, becoming paralyzed. A striking feature of the intranasal inoculation method is the regularity of the incubation period in groups of monkeys treated identically. As the result of the instillations, symptoms of infection, among which is fever, which frequently marks the onset of the disease, often arise on the same day. In this respect there is striking correspondence with the multiple cases observed among children in a family or similar group. We have described the phenomenon under the term "simultaneity of infection."[23] While this phenomenon has been observed chiefly in monkeys receiving virus intranasally, we have found it also in groups of monkeys inoculated at one time intracerebrally.

During the past twenty years not a few observations have been made on human beings, which bear out those made on experimentally infected monkeys. The virus of poliomyelitis has been detected in washings from the nose and throat of the parents of paralyzed children,[24] in unaffected children in contact with paralyzed brothers or sisters[25] and in children suffering from the indefi-

nite, minor illnesses believed to be poliomyelitis, in communities in which the typical disease was occurring.[26] The virus has in one or two instances been detected in healthy children, under the circumstances related, and these "carriers" have themselves fallen victims to the paralytic disease a few days later.[25,26] Moreover, tonsils removed from patients in the course of the disease, or from fatal cases at autopsy, have been shown to harbor the virus.[27]

The introduction of virus into the gastrointestinal tract rarely leads to paralysis. In the instances in which paralysis does occur, the question arises whether the virus may not have come into intimate contact with the nasopharyngeal mucosa. The substitution of Macacus cynomolgus for Macacus rhesus, as recommended by Levaditi and his associates,[28] does not change the results when rigorous experimental conditions are maintained. It is possible to compare the effects of virus instilled into the nose of monkeys which have resisted the feeding of virus, with the lack of effects obtained in feeding experiments. For this purpose, specimens of the same adapted virus were employed.

It will be recalled that 6 cynomolgi were given successive virus feedings by stomach tube. Five remained normal, while the sixth developed a nonprogressive paralysis of one leg. The 5 resistant monkeys were then given 6 daily nasal instillations of virus. Three of the 5 promptly became infected and succumbed to progressive paralysis; 1 developed abortive symptoms and recovered; the remaining animal, as well as the control monkey, was unaffected.

This experiment is highly informing. By its means, a precise comparison has been made of the vulnerability of the gastrointestinal and the nasopharyngeal mucosa toward the virus, with an obvious, unequivocal result.

It is proposed now to turn briefly to another question, an important one, relating to the nasopharyngeal persistence of the virus. The question concerns not only the occurrence of carriers in general, of which we have evidence in both man and the monkey, but of chronic carriers. The experimental determination of carriers is not easy, since tests must be made in most instances with the use of diluted washings. Such tests made of washings from recovered monkeys, in which passage adapted virus had been employed for inoculation, are likely to be more successful than tests made with human washings, inasmuch as the virus of man has a low initial infectivity for monkeys. Yet both tests have been accomplished

successfully, and after considerable intervals—up to six months—following an acute attack of either the experimental or the human disease.[29] We must, therefore, bear in mind that chronic carriage of virus in poliomyelitis may in part be responsible for appearances or reappearances of the endemic or epidemic malady.

One other matter of importance may be touched upon lightly. Until recently all virus strains, whether secured in Europe, America or elsewhere, have been regarded as being immunologically identical. This was a natural point of view, since convalescent human serum seemed to neutralize all the strains, irrespective of their places of origin. Later facts, however, seem to point in another direction. A strain of poliomyelitis virus secured during an epidemic in Victoria (Australia)[30] proved antigenically different from the old Rockefeller Institute strain. All the evidence since secured upholds the observation of Burnet and Macnamara. It is admissible to go even further and to state that the passage of a strain through monkeys modifies its immunological reactions. As the strain becomes adapted to the monkey, it reacts quantitatively in a different manner to human convalescent serum.[31]

Another difference among virus strains should be mentioned. The strains obtained in a given epidemic year from nearby places show pronounced and permanent variation in potency. A recent 1932 Philadelphia strain proved far more infective for monkeys than the 1931 New York strain which has retained a consistently low activity; but a second 1932 Philadelphia strain has proven less potent than the first one isolated. No correlation has thus far been made between activity for monkey and man; in this respect the virus strains simply repeat what is already well known with regard to strains of bacteria.

Poliomyelitis as a Respiratory Infection. The observations presented seem to favor the nasopharyngeal mucous membrane as the site from which the virus penetrates into the body, reaching eventually the brain and spinal cord. According to this view, poliomyelitis becomes a form of respiratory infection. Attention has been directed to the anomaly of a widespread respiratory infection arising during the spring and summer season and reaching a peak in the autumn months of the year. In discussing the facts, the occasional occurrence of winter outbreaks[32] of poliomyelitis may be disregarded. It is generally admitted that in ordinary epidemics the greatest number of cases occurs in the Northern Hemisphere

during the early, warm autumn period, and in the Southern Hemisphere during the corresponding period of the year.

Perhaps the doubts will be rendered less disturbing if a comparison is made of the conditions under which infection is believed to arise in poliomyelitis and the more common respiratory diseases. In poliomyelitis, as far as is known, no lesions are produced in the nasal mucous membrane, and no symptoms purely respiratory in character arise. There is a tendency to think of upper respiratory infections as associated with obvious lesions and symptoms in the tract itself. The difference in poliomyelitis probably depends on the manner in which the virus acts on the membrane. In contrast to the usual incitants of respiratory infections, it does not attack and injure the membrane, using it merely in order to reach the nerve filaments imbedded in it, along which it passes to the nerve centers and apparently to other parts of the body also, since so many cases of undoubted poliomyelitis develop no pronounced nervous symptoms whatever. The latter is a moot point, since the virus has been detected in so few locations in the body. The indications are that the virus is transported along the axis cylinders; it has, however, shown itself capable of surviving, perhaps vegetating, also in the nasal mucous membrane. While the point is still not definitely determined, there are indications that the virus utilizes the normal or intact mucous membrane more readily and easily as a means of penetration into the body than the injured or inflamed one.

Prevention. The investigation of the immunological phenomena in poliomyelitis has become a rewarding field of experiment. The knowledge of the phenomena has become considerable during the more than twenty-year period of the experimental study of the disease, and the application of this knowledge to the prevention of epidemic poliomyelitis has met with encouraging results in the severe outbreaks occurring in New York State in 1931 and in Pennsylvania in 1932. I shall endeavor to guide you quickly through the main discoveries which have led to the practical achievements to be described. It is, however, necessary at the outset to explain that I have taken the lecturer's privilege of including in this statement certain later results bearing out the earlier ones presented, which were obviously not available when the lecture was delivered. It is sometimes advantageous to defer writing a lecture until the time of publication arrives, especially when, as

in this instance, a new method has been under trial. During the intervening period the method may have been given a wider test, with results sometimes favorable and, of course, sometimes unfavorable to its employment. We appear in this instance to be in the happier situation, and while it is still too early to pass final judgment on available means of preventing poliomyelitis in the young during the prevalence of an epidemic, it is desirable that the nature of such means shall become widely known, since epidemic poliomyelitis continues to appear annually in some parts of America and Europe during the summer and autumn season.

The observation of Flexner and Lewis,[33] that monkeys which had recovered from an attack—irrespective of its severity—of experimentally induced poliomyelitis were not subject to reinfection, led quickly to the testing of the blood of recovered monkeys and human beings for immune substances to which the protection might be attributable.[34] Tests made almost simultaneously in France, Germany and the United States disclosed the existence in the blood, after recovery from the disease, of neutralizing, antiviral bodies. A mixture consisting of the virus of poliomyelitis and the serum of the blood was injected into monkeys. No symptoms of disease tended to arise from this injection; while mixtures of virus and normal monkey serum, or the serum of many but not all persons not known to have had poliomyelitis, proved incapable of protecting the animals against the onset of the symptoms, including paralysis, characteristic of the experimental disease. I shall return a little later to a consideration of the circumstances under which the blood of supposedly normal individuals acts in a measure similar to that of persons known to have had paralytic poliomyelitis, since this action has come to have such pregnant meaning.

Let me repeat, the blood of normal monkeys invariably failed to act upon or neutralize the virus of poliomyelitis. The neutralizing property was detectable only when the inoculated monkeys had shown unmistakable signs of infection, although these signs may have been fleeting in character and wholly devoid of a paralyzing effect. Monkeys which receive the virus in the ordinary process of inoculation and resist all infection—either because the virus employed is too weak, or too small a quantity is used, or because individual monkeys are exceptionally refractory—remain normal in regard to the antiviral action of the blood and capacity to respond with paralytic symptoms when a more effective inoculation is made.

In the light of the fact that monkeys are not naturally afflicted with poliomyelitis, as is man, it is significant to note not only that normal monkeys do not exhibit the blood antiviral property, but that the blood of recovered monkeys is weaker in antiviral substances than is the blood of recovered persons. For the sake of convenience it is customary to speak of the neutralizing, antiviral blood serum obtained from recovered persons and monkeys as "convalescent serum." Thus, monkey convalescent serum is less potent than human convalescent serum. But the potency of the former can be markedly increased by the injection of additional virus into recovered monkeys, a process which Flexner and Lewis called "reinforcement."[35] Reinforced (or hyperimmune) monkey serum equals or even surpasses in neutralizing power the convalescent serum obtained from human beings.

Neutralization of the virus by convalescent serum, monkey and human, was established originally by test-tube experiments. As already stated, virus and serum were mixed before the mixture was injected into monkeys. Since under such conditions no infection occurred, the immediate question which arose was whether neutralization could also be effected inside the animal body, that is, when virus and convalescent serum were injected separately. While the test-tube, or *in vitro* demonstration, of the presence of antiviral bodies in the blood undoubtedly threw light on immunity in poliomyelitis, it was thought that the determination of neutralization *in vivo* might possibly lead to therapeutic application. The tests carried out by Flexner and Lewis[36] consisted of the intracerebral inoculation of virus and the intraspinal injection of convalescent serum into monkeys. The intracerebral method is the most effective that is known for inducing the infection; the intrathecal injection of serum is the most certain and efficient procedure for bringing the neutralizing serum into intimate relation with the nervous tissues, including the nerve cells, and hence for the blocking of the cells against the entrance of the virus, the injurious effects of which are responsible for the severe symptoms of the disease. While it is true that under physiological conditions the flow of the cerebrospinal fluid, into which the serum is introduced, is away from the nerve tissues, the small rise in pressure produced by the injection suffices to reverse the current, bringing the serum into intimate contact with all the constituents of the tissues, including the nerve cells.[37]

The results of the *in vivo* experiments can be stated briefly. When the virus is injected not longer than from eighteen to twenty-four hours before the serum, and the amount of virus introduced into the brain does not exceed a fixed quantity, neutralization *in vivo* can be accomplished. Neutralization may be complete, when no symptoms whatever arise; or it may be partial in which instance mild symptoms appear after a longer incubation period. Ordinarily, the experimentally produced paralytic disease in the monkey is severer than the paralytic disease in man. While the mortality of the human affection is 20 per cent or less, that of the frankly paralyzed monkey is 60 per cent or more. When monkeys recover at all, they have, as a rule, merely passed through a mild attack of the experimental disease, such as is produced with weak virus strains or after partial neutralization of the virus. The *in vivo* experiments showed, therefore, that the action of the virus already present in the animal body could, under certain conditions, be suppressed. Suppression is, however, possible only when the immune serum is injected before symptoms of infection arise; once signs of infection are present, the serum is incapable of preventing the ordinary course of the disease from supervening.

The test by intracerebral inoculation of the virus is the most drastic one that can be devised, exceeding in severity the conditions of infection occurring in man. In the human being the virus is believed both to enter and to leave the nervous system by way of the nasal mucous membrane; Flexner and Lewis[6] early found that the virus implanted in the brain escapes into the nasal mucosa. This observation was quickly followed by the induction of the experimental paralytic disease through the instillation of the virus into the nares,[38] which finding in turn soon gave rise to the discovery that the intrathecal injection of serum prevents the development of infection by way of the nasal membrane.[39] It has, indeed, proven easier to prevent infection by the nasal than by the cerebral route of inoculation.[40] An important consideration is that the time interval between the nasal instillation of virus and the intrameningeal injection of serum may be lengthened with security beyond the period of safety that has been determined when the virus is inoculated into the brain. Moreover, convalescent serum injected into the meninges is equally capable of preventing infection by virus introduced into the blood;[41] and the immune serum is also protective, under certain circumstances, when the injection is made into the

blood stream instead of into the membranes surrounding the brain and spinal cord.[42]

Professor Netter, of Paris, in 1911,[43] seized upon these experimental results and introduced into practice the convalescent serum treatment of poliomyelitis, which has since been the subject of so much discussion. It soon became apparent that, once paralysis had appeared in human beings, the serum was quite powerless to influence the disease. As means of diagnosis became more accurate and instances of preparalytic poliomyelitis could be diagnosed with greater certainty, the use of the convalescent serum was increasingly restricted to these early cases. Medical opinion is still divided as to whether the serum is effective even under these circumstances. The matter has, indeed, become largely a statistical one, and hence it is not likely that a definite decision will be reached immediately.[44] Epidemic poliomyelitis is a protean disease, symptomatically considered, and its effects are so varied that the outcome of individual preparalytic cases cannot be predicted with certainty. Even the comparison of alternate cases in which serum is given with those from which it is withheld is at best but a rough measure of effectiveness, since so much may depend on the way in which the choice of cases is made. I have attempted to formulate my own views[45] on this debated subject, which I venture to restate here:

"My own experience has been chiefly with the experimental disease in monkeys. There the use of immune serum does make a difference. As between human beings and monkeys, the conditions are not identical; but the advantage is in part with human beings. Among them we find far more of the mild or preparalytic cases. In man there is, therefore, an inherently effective agency at work in aborting poliomyelitis. This agency is far less effective in monkeys in which the experimental disease tends to be severe and fatal. The severe form of the experimental disease in monkeys can be prevented or mitigated by means of immune serum. Since the practice of medicine is applied to individual cases of disease and does not, except statistically, deal with disease in mass, the question may fairly be asked whether the preëxisting mechanisms in man tending to confine and abort the virus infection cannot in some instances be supplemented and fortified by convalescent serum. It is generally agreed that the use of the serum does no harm. Since it cannot be affirmed that in individual cases it does no good, and a body of medical opinion exists in its favor, the question arises

whether its use should be withheld. This question is to be answered not by the pathologist, but by the physician. If a better, more calculable method of preventing paralysis were known, that is, a surer way of supplementing the normal mechanism tending to restrain the action of the virus before it causes functional injury to the nerve cells, this question would not arise. But there is no better or more calculable method known. The choice is, therefore, between no therapeutic intervention at all and a kind of intervention believed to be harmless and not known actually not to be sometimes useful in cases of this tragic disease."

Up to this point we have dealt with the restraint of the virus when it is mixed with immune serum in the test-tube, or when the virus introduced into the animal body is followed by the injection of serum before symptoms of infection have arisen. The next problem to be considered relates to the possibility of restraint of the virus when the serum is injected several days in advance of the virus. The answer to this question received from experimental work promises to bear on a possible preventive measure—passive serum protection—against epidemic poliomyelitis.

Flexner and Stewart on addressing themselves to the solution of this problem found that serum prevention is not only experimentally possible, but that the blocking effect against the entrance of the virus into the nerve cells endures for several days after an intraspinal injection, and occurs even after an intravenous injection of convalescent serum. Having in mind the practical implications of these observations, they proposed "that in the event of severe outbreaks of epidemic poliomyelitis, convalescent human serum be employed to afford passive protection to persons—children especially—menaced by the disease."[42]

The experimental results having been successful, we regarded it desirable to apply the method of protection to children and young adults exposed to epidemic poliomyelitis. During the next two or three years, of a number of such persons injected with convalescent serum, none developed the disease; but the number was too small to permit decisive conclusions being drawn. An opportunity came, however, with the overwhelming New York City epidemic of 1931, at which time I recommended the protective injection of convalescent serum or its near equivalent, blood from normal adults, parents by preference.[46] The amounts of convalescent serum available are too limited to serve for mass immunization. Several thousand

children were treated in this manner, and among them the incidence of poliomyelitis was believed to be lower than in the uninjected population. The procedure employed was to withdraw, from parents, blood taken under sterile conditions and to inject 30 cc. or more of the uncoagulated blood intramuscularly into the children.

Allusion has been made to the fact that the blood of normal adults offers a substitute for the convalescent serum. The history of the discovery of the virus-neutralizing property possessed by the blood serum of normal adults on the virus of poliomyelitis is instructive. In the year 1911, Anderson and Frost[47] investigated the so-called "abortive cases" of poliomyelitis to which Wickman's attention had been forcibly directed in the Swedish epidemic of 1905–1906.[2] The American investigators paid particular attention to the blood-neutralizing reaction which had been described a short time before,[24] by the employment of which they endeavored to establish the poliomyelitic nature of certain indefinite cases of illness which accompanied those of frank paralysis arising in the epidemic of poliomyelitis at Mason City, Iowa. In the course of their studies they made two observations, since confirmed, which are of special significance: (1) That the blood of adults who had shown no signs of illness may possess considerable antiviral properties; (2) that the blood of young children, even when they have had an attack of the disease, may not promptly develop neutralizing power against poliomyelitis, or may develop it in a lesser degree than adults. Similar observations were made by Peabody, Draper and Dochez,[48] who studied the New York City epidemic of 1911.

But it was Aycock and his associates[49] who studied the phenomenon more widely and concluded that a process of unperceived, subclinical immunization of the adult population is taking place extensively today, thus explaining the antiviral activities now so generally found in the blood of normal persons. Aycock relates this subtle process to the similar one long known to be occurring in widely prevalent infectious diseases such as diphtheria and scarlet fever. Normal adult blood not only tends to contain the neutralizing substances in appreciable amounts, but in rare instances in quantities surpassing those present in the blood of persons who have recovered from undoubted attacks of poliomyelitis.[50]

However, it was not until the epidemic of 1932 in Philadelphia and other places in Pennsylvania that the method of passive immunization by the use of normal adult blood received not only wide,

but more measured application. The analysis of these tests is being awaited with great interest. Certain favorable results have already been reported. But the completion of final results, and especially their interpretation, will require time and critical judgment. It is obvious, of course, that comparison should be made, where practicable, between large groups of children, injected and uninjected, both groups being similarly circumstanced as to age, environment and exposure. But it is also desirable to take into account the fact that certain adults are known not to possess the antiviral blood property; hence a standardized protective material cannot be generally employed in the effort to produce passive immunization.

Finally, it must be remembered that tests on monkeys have disclosed two modes of intravitam action of the convalescent serum. In one, the virus is completely suppressed and no symptoms of infection arise; in the other, the action of the virus is mitigated, the incubation period before the appearance of symptoms being prolonged, and the symptoms themselves are milder. It is probable, therefore, that among the passively immunized children many will escape all signs of disease, and others—depending on all the circumstances stated—will fail to be protected, or will develop milder symptoms, taking into account the number receiving the adult blood in comparison with a corresponding number not so treated. We must await future events before attempting any general prediction on this important subject; but enough would appear to have been achieved already to warrant a further and wider trial of this safe and readily available means of preventing epidemic poliomyelitis which continues to be a serious menace on a world-wide scale.

One aspect of the subject of immunity in poliomyelitis remains to be considered. We have long known that monkeys may be rendered actively immune by successive small injections, or one large injection, of poliomyelitis virus made beneath the skin,[51] or by successive inoculations made into the skin.[52] Both these methods of securing immunity suffer from the disadvantage that occasionally monkeys develop paralysis instead of immunity. It appears that monkeys, in common with human beings, exhibit varying degrees of susceptibility to the presence of the virus in the body. An effort is being made to improve and perfect this means of active immunization in order to avoid the rare onset of disease. The com-

bined use of immune serum and virus offers greater security. That active immunity can be obtained by the injection of mixtures of the serum and virus has been shown by Rhoads.[53] Recently we have carried out on a larger scale experiments in which virus and serum have been injected separately into the body of monkeys. These animals developed active immunity and, up to the present, without the appearance of symptoms of infection in any case. The possible application of a method of active immunization to the prevention of the disease in man is a subject merely to be alluded to at this time.[54]

In concluding this address, I should like to add that although epidemic poliomyelitis is still an all too common disease and its effective prevention is still to be attained, yet the knowledge of it that has been gained during the past quarter of a century, mainly through the employment of the experimental method of investigation, is considerable. It is, therefore, to be hoped that with the further growth of knowledge, a greater measure of practical control may be secured.

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PREVENTION OF DEFORMITY IN INFANTILE PARALYSIS*

By DeForest P. Willard, M.D.

Although more than one hundred and fifty years have past since the disease entity we now know as infantile paralysis was first described, it was not until 1900 that its infectious character was recognized. Since that time great advances have been made in our knowledge of its etiology and pathology, and this advance we owe in a very large part to the brilliant investigative work carried out at the Rockefeller Institute under the leadership of Dr. Flexner. As our knowledge of the disease increased so has our ability to combat, to curtail and to correct its ravages increased. As in all diseases, our first forward strides were in the correction of its more obvious deformities; but in the past decade we have also made a good beginning in the more important department of treatment, the prevention both of the disease itself and of the disabilities that arise from it.

To entirely prevent its deformities we must eradicate the disease, and in this the research worker and the clinician have joined forces. We now know that it is an infectious disease due to a specific virus and we also know that one attack gives a marked immunity. With these basic facts we can confidently predict an increasingly efficient attack by the use of preventive and curative sera.

Already human blood serum especially from convalescent patients has proved most useful when used in the preparalytic stages. In our own hospitals during the past summer we have had several undoubted cases of infantile in which the paralysis has been either markedly lessened or entirely prevented. This type of serum is of necessity difficult to obtain and expensive to prepare and we have yet to obtain any type of serum that can be produced in large amounts.

At the present time we are ignorant as to the precise methods of transmission, but when this stumbling-block is overcome, as it will

* Read January 6, 1932.

be some day, we will no longer have to fear the disastrous epidemics that have left so many physically handicapped children in their wake.

Even when the disease has developed in an individual we still can do much to prevent deformity by mechanical appliances, and upon the stage of the disease depends the type of treatment. It is customary to divide an attack of infantile paralysis into three stages. (1) The acute stage, from the onset to the disappearance of deep tenderness along the nerve trunks, with a duration of two to twelve weeks. (2) The convalescent stage, from the disappearance of tenderness through the period of recovery of motor functions, with a duration of one to three years. (3) The chronic stage of permanent paralysis. It is in the first two stages that we may hope to prevent deformity.

In the beginning of the acute stage we are dealing with a patient who is seriously ill. As the disease progresses the symptoms of an acute intoxication decrease, but signs of paralysis and painful neuritis increase. Our patient is, therefore, confined to bed throughout this entire stage and our indications for treatment from the preventive standpoint are the restriction of the infective process and the complete immobilization of the affected areas in correct position. In the early paralytic, or preparalytic, stage much has been done and more may be expected by the use of serum. Even when paralysis has once developed we may still prevent deformity. These deformities are due not to the paralysis of muscle groups, but to the uncontrolled pull of unparalyzed muscles which would normally be counterbalanced by the paralyzed groups. The pull of gravity may often assist the stronger muscles in bringing and holding the limb in an unnatural position. For example, the most common deformity is equinus or foot-drop. It is caused by the overaction of the calf muscles and the underaction of their opposing muscles, the dorsiflexors, plus the pull of gravity on the foot itself. At first this position can easily be corrected, but in a very short time the muscles become set in the abnormal position and reposition becomes exceedingly difficult or even impossible.

As soon as the paralyzed muscle groups have been diagnosed, it is, therefore, necessary to hold the affected part in the neutral position so that the strong muscle groups do not become permanently set in the contracted positions. Splints, sandbags and plaster casts are most commonly used to maintain these correct positions

and to keep the limb immobilized during the painful stages. These are all temporary types of apparatus and the choice of these various methods depends on the needs of the individual case.

In the convalescent stage, when the amount of paralysis has been well established and the work of rehabilitation has been started, preventive appliances are as equally important as in the acute stage. Here the picture changes. We are now dealing with a patient who is able to bear weight with assistance and who now must be aided in exercising the weakened muscles without allowing them to become exhausted. Now it is necessary not only to keep the strong muscles from contracting and to prevent the weakened or paralyzed muscles from being overstretched, but to continue such treatments over a long period of time. It is a well-known fact that no overstretched muscle will regain its normal power while it is in a position of over-extension. Moreover, it is of great importance to see that the correct position is maintained during the entire twenty-four hours of each day. So often the good obtained by holding the affected limb in correct position by braces during the daytime is entirely neutralized by allowing it to fall into the contracted positon during the night. The types of apparatus that may be used to prevent deformity are splints, casts and braces. Splints are most useful in maintaining correct position in the upper extremity where the pull of the muscles and of gravity are not great. Plaster-of-Paris, in the form of a full or half cast, is most commonly used in the lower extremity where the forces of muscle and gravitation pull are more severe and where weight-bearing without muscle action is indicated. Both splints and casts may be used as night appliances in connection with braces. Braces of various materials are probably the most commonly used form of apparatus. The brace of today is very different from the cumbersome appliance of a few years ago. The lighter metals, such as duralumin, have taken the place of steel and leather, and have enormously diminished the excessive weight. Celluloid, especially in the prevention of back deformities, is now extensively used on account of its extreme lightness in place of metal braces or plaster-of-Paris casts. Braces are mainly used where support must be combined with locomotion. Their lightness is, therefore, an essential factor, especially in small children. They must fit snugly. They must give rigidity where weight-bearing is essential and yet must allow motion at the joint lines. By the addition of elastic straps, braces may be made to reinforce the action of

the weakened muscles. They are, therefore, the most complicated pieces of apparatus that can be used in the prevention of deformity, and every small detail of their make-up must be carefully thought out. It is never fair to the patient nor his doctor to ask the brace-maker to assume the responsibility of correctly making and fitting such an appliance and it is distinctly up to the physician in charge to see that his ideas are correctly carried out.

Surgical procedures play a very minor rôle in the preventive treatment. Occasionally, where the various forms of apparatus have been unsuccessful or where early deformity has occurred, manipulation under anesthesia, subcutaneous tenotomies or tendon lengthenings may be necessary, but they are only used as adjuncts or aids to the various forms of mechanical appliance.

A detailed description of the various methods used in the prevention of the individual deformities due to infantile paralysis would fill a large-size volume and would be entirely out of place at this time. Only the most general principles have been touched upon, but the importance of the principles cannot be too heavily stressed. Briefly stated, they are: (1) A greater knowledge of the etiology and modes of transmission. (2) The use of serum in the early stages and the better and more extensive preparation of such serum. (3) After the disease has been established, the early and careful application of mechanical appliances to overcome the unbalanced muscular pulls and to assist in muscle function when muscle power is returning in the paralyzed groups. The closest coöperation between physician, surgeon and parents is absolutely necessary to obtain the best results in the long fight against the physical defects caused by this disease. But if this teamwork be efficiently maintained, and if year by year our research colleagues supply us with new and better armament, we can be assured that, before long, infantile paralysis will be classed among the diseases that medical science has conquered.

THE PURPOSES OF THE COMMITTEE ON THE COSTS OF MEDICAL CARE*

By ARTHUR C. MORGAN, M.D.

The committee on the costs of medical care was created to study the problem of "the delivery of adequate scientific medical service to all the people, rich and poor, at a cost which can reasonably be met by them in their respective stations in life." (Olin West, Secretary, American Medical Association.)

The problem of the economics of medicine has been informally discussed for many years. The time seemed ripe to have a series of studies made in order to evaluate this important subject. This led to formation of a committee composed of Winford H. Smith, M.D., Baltimore, as chairman; Mr. Michael M. Davis, of the Julius Rosenwald Fund; Walton H. Hamilton, Ph.D., Yale University; C.-E. A. Winslow, doctor of public health, and Lewellys F. Barker, M.D. H. H. Moore, Ph.D., holds the important office of secretary. The first general conference was held in Washington, May 17, 1927.

The committee as now organized consists of 16 representatives in private practice of medicine and dentistry; 6 of public health; 12 of institutions and special interests; 6 of social sciences and 10 representing the general public. In addition, there is a research staff of 12 persons.

Ray Lyman Wilbur, M.D., secretary of the interior, is chairman of the general committee, while Dr. Winslow is chairman of the executive committee.

Pennsylvania is represented by Ambrose Hunsberger, Phar.M.; Morris L. Cooke, D.Sc., management engineer, and Arthur C. Morgan, M.D. Recently David Riesman, M.D., has been added as consultant.

The work is now in its fifth and last year and is rapidly approaching a point where opinions may be expressed upon data compiled and beginning crystallization of sentiment that will be shown in the final report of the committee which, it is expected, will be published early in 1933.

* Read February 3, 1932.

The general committee was obligated to approach the study with unbiased minds, discarding any hard or fixed opinions that may have been entertained by its individual members, so that when the final conclusions are presented they will represent the majority opinion of the committee. It is possible that minority opinions may be expressed by some individuals, but on the whole it may be stated that the general committee is working as a unit toward the accomplishment of its worthy purpose.

Trained workers have studied the medical economic situation in Germany, England and Austria. A cross-section of Canada has been studied. The scope of the study includes the economic aspects of physicians, dentists, nurses, hospitals, midwives, chiropodists and optometrists, pharmacists, cults, patent medicines and the like, in an endeavor to approach the study from every possible angle.

Considerable publicity has been accorded to the work of the committee, favorable and critical, as it was felt proper "to educate the public mind with an attitude of expectancy toward the publication of its final report."

Some objectives that have been pursued are "to teach people (1) that all of the sick are not well cared for; (2) that payment of medical costs is not possible for some and not satisfactory for others; (3) that fertile minds and ingenious administrators are trying to meet these problems, and (4) that the people as a whole and as members of various groups have definite responsibilities to help in the solution of these problems and to put the committee's recommendations into effective action."

Printed reports are already available covering twelve completed studies. Bulletin No. 9, "A Survey of the Medical Facilities of the City of Philadelphia, 1929," is of special interest to medical Philadelphia because of its local application and home city study. The writer will endeavor to secure a complete set of publications, abstracts and final reports of the committee for the College library. Publications may be obtained from the University of Chicago Press, 5750 Ellis Avenue, Chicago, Illinois.

Possible application of the insurance principle to payment for medical care is being studied by the committee. A recent study of this phase of the study summarizes: "Insurance companies estimate that $3,600,000,000 annually is invested in life insurance and, in addition, another $600,000,000 a year is deposited sys-

tematically in the various Christmas saving funds. The committee has shown that more than $3,000,000,000 a year is spent throughout the country for medical care. The first two sums result from regular systematic payments and budgets, whereas the latter enormous sum represents a hardship in that little or no provision is made by individuals or families for a "rainy day."

Other angles of study have been as follows: Private group medical and dental service; "pay" clinics for diagnosis and treatment; middle-rate plans for hospital patients; visiting nurse service; industrial and university medical services; voluntary group-payment agreements for annual medical service; local coördinating health agencies; county medical society clinics; salaried community physicians in rural areas; comprehensive community medical service centers. At all times the committee has kept in mind throughout each study the keystone proposition that the family physician is the basic unit in medical care.

The summary of Study 9 shows: "In Philadelphia in 1928 the percapita expenditure for medical care was $53.89. An appraisal of Philadelphia's public-health activities, according to the American Public Health Association standards, gives a total weighted score of 73.8 (on the basis of 100). The city makes high scores in the fields of maternity, hygiene, communicable-disease control, heart-disease control and infant hygiene, but low score for sanitation (including the provision of potable water and the sanitary disposal of sewage), preschool hygiene, cancer control, venereal-disease control and school hygiene. The city's hospital facilities have apparently developed without effective community planning, as there appears more than adequate bed capacity in general and special hospitals for acute illness but a dearth of facilities for nervous and mental, tuberculous, convalescing and chronic patients. The follow-up service among patients discharged from hospitals seems to be inadequate and some institutions completely lack social service.

"There are no accepted standards by which to gauge dispensary facilities, but it is apparent that preventive services are not extensive. Philadelphia had 6 per cent fewer physicians per 100,000 persons in 1928 than in 1906. Most Philadelphia physicians, especially general practitioners, work long hours and this may indicate a present inadequacy. Specialists are numerous, perhaps too numerous for the best interests of the city.

"Most Philadelphia residents are not receiving dental service according to the highest professional standards, perhaps in part due to their unwillingness to pay.

"The family of average size expends approximately $200 to $250 per year for medical care. Comparison with other cities must await additional data from formulation of suitable standards.

"It may well be asked whether or not, in the very multiplicity of agencies and individuals concerned in the provision of medical care in Philadelphia, there is an outstanding weakness of the present system. Many individuals and agencies of the same general type operate without effective coördination, and this lack of coöperation is even more striking between different agencies or groups. To the end that each medical facility may render its greatest usefulness to the public and coöperate most effectively with all the others, Philadelphia apparently needs to coördinate the agencies concerned in its percapita expenditure of $53.89."

No attempt has been made by the writer to expound personal ideas or recommendations, attention again being called to the obligation agreed to by the members of the committee not to assume any final opinions or conclusions until the completed report is in the hands of the public.

"It is the committee's hope that the facts brought out by the various studies will make possible, at the completion of the program, a report summarizing results with recommendations for the provision of more efficient medical service for all the people, not only rich and poor, but also persons of moderate means." (*From Foreword to the Five-year Program.*)

SOME NOTES ON SOCIAL INSURANCE AND HEALTH WORK IN GERMANY AND AUSTRIA*

By HENRY FIELD SMYTH, M.D., Dr.P.H.

That which I have to present this evening does not justify the name of "paper." It is compiled from a few notes taken during personal interviews with Dr. Thiele in Dresden and Dr. Sherer, director of health of Vienna, in Vienna, the former discussing the working of the German social insurance program and the latter the activities of the health department in a large city under a strong social government. These notes are perforce incomplete, as they were made for my own information and future reference, with no thought of their forming the basis of a paper or other formal communication.

German social insurance is administered under a panel system, all employees earning over 3600 marks (or $504) per year being insured compulsorily, the insurance covering accident, health and employment. This provides medical treatment and medicines during illness, and hospitalization with money for family support, or else 50 per cent of the wage. Childbirth care is also provided for and financed, and funds are provided for the care of the family during the period when the mother is incapacitated.

The fund is maintained by contributions totaling 16 per cent of the wages of each insured individual, of which the employer pays 7.57 per cent and the worker 8.43 per cent. The worker may increase the amount of his benefits by voluntary contributions.

In 1930 there were in Germany over 22,000,000 insured workers, of whom 54.9 per cent received treatment during 1929. Agricultural workers applied for treatment on an average of once every third year, and industrial workers once every second year, while miners applied four to five times a year.

There is one panel doctor for every 1000 insured, and during 1929 they treated 40 per cent of the population of Germany. The panel doctor's income averages 11,000 marks ($2750) per year.

* Read February 3, 1932.

At the time this information was obtained there were 3,000,000 unemployed workers in Germany receiving some aid from the fund.

Dr. Sherer, in a talk to a group of public-health workers gathered in Vienna, gave us some very interesting information as to the activities of the health department. Vienna is a city of 1,800,000 inhabitants, and one of ten Austrian states. At present it is under a Socialist administration, at the last election about 75 per cent of the vote being socialistic.

The city health department activities are divided among seven divisions or bureaus. The activities of the divisions of hospitals, infectious diseases and sanitation are similar to those of an American department. In addition, there is an employment division, a social service division and a children's health division, the activities of which are of especial interest to this group. I have no figures as to the activities of the employment division. The social service division renders aid to adults and at the time there were 40,000 monthly pensioners being cared for in whole or in part, 112,000,000 shillings ($15,680,000) being spent annually for this service.

The child health bureau activities are most interesting. The city is divided into 100 districts for health work. All pregnancies and births must be reported and a free layette is supplied for babies of the unemployed. Pregnant women may receive aid twice monthly, provided they report within the first four months of pregnancy. At the time, 16,000 families were being so aided.

All children, regardless of social or financial status, are examined by department representatives monthly. If found defective, backward, undernourished, sick or in bad moral surroundings, they are sent for ten weeks to an observation center, where they are observed and studied from all angles. This is a beautifully kept and appointed institute, which we had the pleasure of visiting. The children live in cubicles in small groups, or at first in separate rooms, if advisable, and they receive the best of medical, nursing, health and educational care.

After the observation period is over they may be:

1. Sent to a hospital or sanitarium for treatment at the expense of the state, if necessary.

2. Sent to foster parents who care for them at the expense of the state and under regular departmental supervision.

3. Sent back home, with state aid if needed.

At the time, 20,000 children were being fed a 700-calorie daily lunch at state expense.

The city maintains a very well-equipped and administered bathing establishment, with free plunge, shower and tub baths, for all, and better appointed baths for those caring to pay for them, as well as municipal open-air pools.

They also maintain enormous groups of municipal apartment houses, where citizens may live at very reasonable rates. Single rooms are available for single persons, but cannot be occupied by two. As families increase in size, they must move into progressively larger apartments, so that crowding is impossible. These apartment groups contain playrooms and kindergartens for children, and workers' children are cared for during the day. Play may be indoors, on roof playgrounds, or in protected courts. Central, well-equipped laundries are provided where housewives may bring their own laundry and work with the best of equipment owned by the state.

In fact the state seems to care for and regulate the lives of all of its members in any way unable fully to care for themselves. But in spite of all this, that conditions are not ideal and do not satisfy the needs and desires of all, is indicated by some figures on suicides in Austria, which I found in a news item in a cafe on Saturday afternoon:

In 1928 there were in Austria 2374 suicides—about 38.7 per 100,000 population.

In 1929 there were 2329, or about 38 per 100,000.

In New York City, which has a slightly larger population, the rate for 1923 was 14.2, the highest rate for ten years being 19 in 1915. In Philadelphia, which is approximately the size of Vienna, the rate for 1923 was 14.8.

In Vienna, in 1929, 58.4 per cent of these suicides were males—41.6 per cent being females. In the United States, in 1923, 75.2 per cent were males and 24.8 per cent females.

In Vienna, 7.4 per cent of the suicides were children, and 48 per cent were from twenty to twenty-nine years of age. In New York City, for 1923, only 1.7 per cent were children, and 14.4 per cent between the ages of twenty to twenty-nine years.

21.6 per cent of the suicides in Vienna were in the educated class.

The above figures are a comparison of those seen in Vienna with rates taken from the 1923 mortality statistics of the United States. The particularly sad part of this is that the higher Austrian rates are due largely to suicides of women and children, and young adults generally.

THE PLAN OF THE BRITISH MEDICAL ASSOCIATION FOR A GENERAL MEDICAL SERVICE FOR THE NATION*

By SAMUEL BRADBURY, M.D.

(ABSTRACT)

During the past decade we have heard a great deal about the high cost of medical care and from the criticisms expressed in lay magazines it would seem that doctor's bill and the costs of care are much confused. When carefully assembled figures are examined, it is found that the doctor's income is not unduly large and that his share of the actual cost is scarcely one-third of the total bill. Furthermore, a preliminary estimate of the annual percapita expense for medical care shows it to be between $25 and $30. This is a little less than that for candy, about one-half of what is spent for tobacco, about one-sixth of the automobile expenditure. These expenditures, however, are quite evenly distributed among all families. If inexpensive, they are paid for a nickel or two at a time, and when expensive often in carefully arranged financing schemes. But sickness in a cross-section of humanity is relatively unusual, and in serious form occurs but once or twice in the lifetime of an individual. Unforeseen and unprepared for, it becomes a hardship. In study after study of people of all economic groups it is shown that the great bulk of the total sickness cost of the group is borne by a very small proportion of families.

Another aspect of this problem is seen in the sickness surveys made by the Metropolitan Life Insurance Company among the families of their industrial policyholders, comprising some 630,000 people of all ages in urban and rural districts. About 2 per cent were sick, nearly all of whom were unable to work and one-third had been sick for six months or more. The interesting fact is that 30 per cent had no medical attention of any kind.

Again, in the last report upon hospitals by the American Medical

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Association, it is stated that in the seven years following 1923 the hospital unit of care (the patient day) increased 38 per cent and that from 1921 to 1928 the proportion of the population of the United States attending the dispensaries increased from 1 in 35 to 1 in 18. Undeniably there is a drift of people to hospital and dispensary for their medical care.

Perhaps there is some relationship between these data—the complaints, the numbers of people without care and the drift of people to the dispensaries. Is it not because wages have not risen as rapidly as the cost of caring for sick people?

The influences in the development of increased cost are the growth of specialism and, probably even more potent a factor, the rapidly accumulating array of diagnostic tests and therapeutic procedures now in use. Many of the latter require expensive apparatus which, in order that it earn its cost, must be located where it may be extensively used—in the hospital. A recent rather startling instance is the care of pneumonia. Six or eight years ago the average drug cost for the care of this disease was perhaps $1.00; now with oxygen tent and serum it may be $100 or more. There may be another factor in the hospital drift; it seems necessary in so many instances to have a diagnostic survey made upon patients, usually with several days spent in a hospital bed. Perhaps these people are taught that such examinations should be carried out in all illnesses, that the hospital is the only place to have them done and possibly that the tests, of which so much is made, are more important than the doctor.

The rich man can pay his way, and the pauper has no difficulty, but what of the people between these extremes, of most indefinite upper and lower income limits, who comprise 80 per cent or more of the nation? They have not been trained to provide for these medical expenses. In fact, there is in this country no generally known method by which such contingencies may be provided for except the savings bank.

The British physician has had experiences similar to ours, but the social relationships of medical practice appear to have been brought more forcibly into the open. Through the work of a decade or more the British Medical Association has developed a plan which it is believed will place all sorts of medical service within the reach of everyone who needs it and will provide proper remuneration for the physician. The scope and purpose of the proposal are best described in the introductory paragraphs, as follows:

"During the past twenty years the attention of the public has been directed more than ever before to the subject of the health of the people. The systematic medical inspection and treatment of school children; the national health insurance system; the striking evidence as to the loss of millions of weeks of work in a year; the establishment and the activities of the ministry of health; the devastating influenza epidemics; the experience gained from the operations of the maternity and child welfare schemes; inquiries into the subject of maternal mortality; the increasing interest in the subject taken by the press. All these have combined to make the problem of how best to promote the health of the people one of the most interesting and pressing public questions of the day.

"The British Medical Association has been busily engaged in studying in detail various aspects of the question. It now feels able to piece these together into a coherent and inclusive scheme; to provide a service for every class of the population comprehensive enough to cover the whole field of preventive and curative medicine.

"The Association, therefore, presents the following scheme to the public and invites the fullest criticism. The public may be assured that the architects are putting forward something which they believe to be complete and adequate for its purpose; which they think is financially within the collective means of its prospective users, and which can be added and adapted to the existing structure with great advantage. The plan has the advantage which a great many schemes presented to the public conspicuously lack, namely, that it is in accordance with the beliefs and traditions of the medical profession and would have its whole-hearted support. It is open to criticism, but the Association believes that the essentials cannot be seriously altered if the public desires to have a scheme which is comprehensive and which will be satisfactory both to the recipients and to the givers of the service."

The plan is built up about eight fundamental principles which cover every kind of (health) service which may be necessary for the prevention and the cure of disease and for the promotion of full mental and physical efficiency.

The first of these principles is "that a satisfactory system of medical service must be directed to the prevention of disease no less than the relief of individual sufferers." As I understand it, one of the chief criticisms of government health insurance, not only by the English physician but by those on the continent, is that these systems make no provision for preventive medicine except through the usual health department procedures. The proposal hopes to accomplish prevention of disease through instruction to the individual in the maintenance of a healthy environment for himself and his family, through the family doctor in the course of his regular curative work among the people under his care; the school by education in matters of health and conduct; health propaganda of the

public-health services and various voluntary agencies and the regularly constituted work of the public health and sanitary services. It is clearly pointed out that progress in prevention of disease is more a matter of education of the people than through direct action by the medical profession. The physician has in the past and will develop the means of prevention; he will provide instruction and encouragement in the use of these preventive measures, but the people must carry out these suggestions. It is stated that there is no real line of demarcation between curative and preventive work, and that, for this reason, every family physician must be closely associated with the general health service. It seems to me pertinent to the subject that the diseases which are prevented by direct action of health officers, such as typhoid fever, are nearly absent from modern communities, while other conditions which depend upon individual action for their prevention are not in like condition.

The second fundamental is "that the general medical service of the community must be based on the provision for every individual of a general practitioner or family doctor." This family physician is to be responsible for the teaching of prevention of disease and for the care of the great majority of ills with which his patients suffer. He is, or should be, particularly valuable to his patients because he is the one who best knows all the factors in family, work and living environment. It is, further, shown that present-day medical care often is a matter of teamwork and that for effective service all parts of the team must work together. This and the desire that the teamwork should be correlated in the family doctor is expressed in the third fundamental, "that a consultant service and all necessary specialist and auxiliary forms of diagnosis and treatment should be available for the individual patient normally through the agency of the family doctor."

They are (*a*) diagnostic aids, such as the consultant and the specialist, including physicians who are skilled in special forms of diagnosis, as pathology, and (*b*) aids in treatment, such as part- or full-time nursing, "home helpers," midwives, masseurs, opticians, physiotherapeutists and pharmacists and institutional accommodations as general or special hospitals, venereal-disease clinics, sanatoriums and convalescent homes. There are, also, advised local "clearing houses" for making readily available various types of information.

It is expected and intended that most of these diagnostic and

treatment aids shall be available to people in their homes. Some apparatus, delicate or unwieldy, cannot readily be transported. When such is necessary it is suggested that there be established at convenient locations "local medical centers" for the use of the family physician, consultants or specialists in the diagnosis and treatment of ambulatory cases.

In payment for medical services the plan carefully explains that the patients will fall into four general classes with respect to financial status:

1. Those able to pay directly for whatever may be required.
2. Those who will be able to pay directly for ordinary domiciliary attendance, but who will find it necessary to use some form of insurance for unusual expenses.
3. Those whose expenses for domiciliary attendance will come under the present national health insurance, but who must pay for institutional care through a voluntary insurance plan.
4. People who must be cared for without any charge to themselves.

For certain classes the service must be subsidized by the state, but it is by no means intended that the entire service will be so supported, and it is expected that it will be largely self-supporting. With respect to the people for whom some sort of insurance is necessary it is held, "that the interposition of any third party between the doctor and the patient, as far as actual medical attention is concerned, shall be as limited as possible." This must be so in order that the sense of personal responsibility be maintained, that the patient feel that the doctor is whole-heartedly acting in his behalf and because such outside interference tends to take a political form and make the system the play of party politics.

In thus extending the insurance it is held, "*that* as regards the purely professional side of the service, the guaranteeing of the quality of the service and the discipline of the doctors taking part in it, as much responsibility as possible be placed on the organized medical profession," and "*that* in any arrangement made for communal or subsidized or insurance medical service the organized medical profession should be freely consulted from the outset on all professional matters by those responsible for the financial and administrative control of the service." These two stipulations appear to be perfectly clear cut—certainly the physician through his own organizations should advise upon, if not entirely determine, professional questions.

The plan urges that any insurance system should be a contributory one for all except the pauper classes. This is now the case with a large part of the population. It is held that contributing tends to promote self-reliance and thrift among the people, and, also, that it will maintain and develop some of the valuable features of private practice such as free choice of physician and an element of reasonable competition. The desire of even poor people to provide for themselves is laudable and should be encouraged; therefore, "*the* medical benefits of the present national health insurance acts should be extended so as to include the dependents of all persons insured thereunder."

The pauper now is cared for by the local poor board. They can as well as not be included in the insurance service, but in their case the entire contribution for care would be made by the poor board directly into the local insurance pool. The amount to be paid is to be determined by averaging up the actual costs of this service during several years past.

Institutional services are considered separately. There are in England two types of hospital, the voluntary and the county or council hospital, which have not competed to any extent. But in 1929 the "local government act" provided that local authorities should furnish institutional care for all who may need it. It is expected that this will greatly stimulate the building of hospitals under local government control, but the act stipulates that the local authority is to confer with the management of local voluntary hospitals in order that resources be pooled and that wasteful competition be avoided. It also provides that local councils are bound to recover from persons treated all or part of the cost of medical care and are authorized to accept payments from insurance funds which are established to provide benefits for members.

The association believed that insurance for institutional care would best be provided by an extension of the national health insurance, but this was found, in 1926, to be impracticable because of the great expense and because so much in the form of contributions would have had to be exacted of many people for whom the services would have been unavailable. It is, therefore, advised that a form of insurance to pay hospital costs be devised which in the future shall also cover payment for the services of the physician. This is one of the most radical of all the proposals. It is explained that the system of free medical service of hospitals is a relic of a former

partnership in charity to the poor, the community furnishing the institution, the doctor the service without charge. But of recent years the criteria for admission have changed from financial to medical needs, and many people admitted today not only can but are willing to pay for their care. If this be done through some form of hospital benefit plan, of which there are satisfactory forms in use in England, a great many of the people cared for can pay. Benefits should not only pay the charges of the institution but also a fee to the physician. Just what this shall amount to is a matter for careful experimentation.

Hospitalization is accomplished because some special diagnosis or treatment is necessary or because the home environment is unfavorable. For the care of the latter type of case the association suggests a special type of hospital of inexpensive construction and equipment in which the family doctor may treat his own cases and in which the consultant and specialist service should be available. The principle is "*that* every effort should be made to provide medical and nursing service facilities in institutions (home hospitals) where the family doctor may treat those of his own patients who need such provision and who can thus remain under his care." These institutions must be conveniently located to the doctors of any district.

Other types of cases will be sent to the larger general or special hospitals on the advice of the family doctor and usually as a result of consultation. As a rule, they will be placed under the care of some particular physician attached to this hospital.

Provision is also made for the institutional care of convalescents, of borderline mental cases, for chronics and incurables and for scientific research and postgraduate instruction, the latter being carried on mainly in the larger general hospitals.

Certain special medical services are provided for. In June, 1929, the Association proposed a scheme for the mother in pregnancy and childbirth, based upon the insurance system and carried out by the family doctor. The claim is made that such services must be considered not in terms of the great cities but rather of the small towns and country districts.

For some time past there has been an infant welfare service. It is held that this should be continued and extended to include the preschool child, but that the type of work should be changed so

that it becomes instruction in "mothercraft," while illnesses or medical conditions should be referred to the family doctor.

The inspection of school children should likewise be continued. It is argued that past experience shows many serious defects found upon the first school examination, defects which have occurred in the hiatus between the well-baby clinic and school age. The school physicians have had considerable difficulty in having these defects remedied, partly due to carelessness upon the part of the parents but mainly because the families are unable to afford the expense. With an insurance service the latter objection will be removed. The association holds that there are certain conditions found in school children which are better cared for in a school clinic because the treatment is carried out by nurses who are thoroughly familiar with these disorders, such as verminous heads and bodies, various types of superficial eruptions and small sores.

The local officer of health is given a very important part in the scheme. He is to be the adviser to the local authority on health matters and it is suggested that he have a duly elected committee of the local profession to assist him in this and in his health-propaganda work. He is to be the administrator for the local institutions which are supported in whole or in part by public funds. He is to be the administrator of such parts of the scheme as need central administration and he is to act as the liaison officer between the local profession engaged in family work and the authority responsible for financing and direction of those parts of the scheme which depend upon insurance and which are provided by the community.

The association indicates the legislation which is necessary to effect the scheme and makes an estimate of its cost. It takes care, also, to point out that a considerable portion of this cost is offset through expenditures of local poor boards, additional benefits under national health insurance, present maternity and child-welfare services, medical and dental examination and treatment of school children and for present types of voluntary insurance.

It is not intended to intimate that such a plan for medical service is suitable for the United States. It is strongly urged that the British Medical Association has observed the needs of the people for medical care, has studied these needs carefully and brought forth and is first in the field with a well-thought-out plan in order that these needs may be met.

A recent article in *Harper's Magazine* is entitled "The Great Fact-finding Farce." It cites a publication of the Russell Sage Foundation in which are listed some 2800 important studies and surveys and it asks very pointedly what has been done about them. The point is this. For nearly five years the committee on the cost of medical care, a nongovernmental organization upon which there are many physicians, has been investigating the subject of sickness and its cost in the United States. This committee will give us the facts, it has already published much important data with which each of us should be thoroughly familiar. If defects in the present organization of medical practice be disclosed, physicians should set about advising their correction. To be first in the field with a satisfactory plan which must fairly consider both sides of the question will give the profession a much stronger position. In any plan of practice it is well to bear in mind the fundamental principles which will make for success and which have been stated by the chairman of the committee:

1. That the personal relationship between patient and physician must be maintained;
2. That provision must be made not only for curative services but for prevention of disease, and
3. That there should be available to everyone all the facilities for diagnosis and for treatment.

THE CURTIS CLINIC: ITS PURPOSE AND ITS POSSIBLE INFLUENCE FOR THE ADVANCEMENT OF COMMUNITY HEALTH*

By ROBERT B. NYE, M.D.

The Curtis Clinic, begun in 1930 and completed in the fall of 1931, opened its doors for the treatment of patients on November 16, 1931. The clinic building was constructed through the generosity of Mr. Cyrus H. K. Curtis and the Board of Trustees of Jefferson Medical College and Hospital.

A Brief Summary of the General Layout of the Clinic. The first floor is occupied by the general admission desk, the drug store, waiting space for patients and the emergency or accident ward. The second floor is occupied by seven offices for the social service department and a much enlarged *x*-ray department equipped with the most modern apparatus for inpatients and outpatients. The third floor is occupied by the Obstetrical department and the Children's department. The specialties—Eye, Ear, Nose and Throat—occupy the entire fourth floor. The fifth floor: the department of general Surgery with its subdivisions—the Varicose Vein clinic, the Follow-up Surgical, the Rectal and the Tumor clinic—and the Gynecological department. The sixth floor is occupied by the Orthopedic and Neurological departments and the Immunology clinic, a subdivision of the general Medical clinic. The Skin and Genito-urinary departments occupy the seventh floor, with a combined department for the treatment of syphilis. The entire eighth floor is taken up by the department of general Medicine with its subdivision, Gastroenterology; the general clinical laboratory for the entire building is also on this floor. The department of Dentistry occupies the ninth tower floor. The basement of the Clinic houses a modern Physiotherapy department for the treatment of outpatients and inpatients. The new emergency ward on the first floor of the Curtis Clinic permits increased facilities for the better care of emergency cases.

* Read February 3, 1932.

The three tower floors, tenth, eleventh and twelfth, house an enlarged department for the nurses' training school, with ample space for lecture rooms, laboratories, a modern library and small study rooms; there are several large demonstration rooms. A very modern dietetic laboratory also adds to the efficiency of this department.

The plan of operation of the Clinic is not entirely an original one, ideas having been gained from the Pennsylvania Hospital, the Johns Hopkins Hospital and the hospital centers in New York City and Boston and adapted to our architectural layout. The first and, to my mind, the most important feature in the plan of operation consists in first determining whether or not the patient applying for treatment is entitled to the services of the Clinic. This is accomplished by means of an interview by the registrar on admission to the first floor of the Clinic. During our short time of operation, we have found that the majority of patients applying for admission are from one of five sources. (1) Refers to the Clinic by outside practicing physicians, some of whom are members of our Staff, others not. (2) Refers from the emergency department for more comprehensive studies. (3) Refers from the general hospital for follow-up care. (4) Patients who are referred into the Clinic by private physicians for an opinion only. (5) Those who present themselves to the Clinic without any previous medical status. From the above summary of the types of patients who present themselves for admission, we feel that the majority of people who apply for treatment are worthy of clinic care. Often we find it necessary to send the referring physician a questionnaire regarding the patient's financial status before admission is thought advisable. This is merely to check up on the patient's statements to safeguard the neighboring practitioner.

The above summary would also give an indication as to the purpose of the organization of such a Clinic. Of course, the present financial depression has increased the number of patients who previously would consult a private physician for medical treatment; on the other hand, there is always a certain group of patients between the stages of the ordinary physician's office fee and complete charity; then there is the usual group of people who are not able to contribute anything toward their treatment. There seems to be a growing tendency throughout the country for patients to contribute something toward the cost of their hospital service.

During the past ten years there has been a more or less gradual and radical change in medical education, and, since Jefferson Medical College Hospital was originally founded for the teaching of its students, the Curtis Clinic provides ample space for the furtherance of this idea. There is a well-equipped classroom in connection with each individual department, and Junior and Senior students of the College are given individual supervision in the care of dispensary patients. This phase of medical teaching could well be summed up in the two words, "preventive medicine." And we find the most important need of such a branch of teaching in all of our medical colleges today, since, after all, the average group of patients who present themselves for treatment at a given clinic represent a cross-section of what the general practitioner sees in private practice. It also affords excellent opportunity for general practitioners by giving a few hours daily.

As mentioned previously, the plan of operation consists of first determining whether the patient is entitled to dispensary care; this interview with the registrar also serves to rate them as free or pay; and if they are found to be able to pay, the economic status and social history determine the fee for clinic attendance. This fee is very flexible, and has to be modified from time to time. The fee is not the cost of medical service, but helps provide modern facilities.

The hospital and outpatient department use a central record room with a unit system of filing records, with a pneumatic tube method of transportation of these records from the record room to the various clinics.

An appointment system has not been generally adopted in all the clinics; a few of the larger clinics use a modified type of arranging visits by appointment. This is particularly necessary in those clinics in which student instruction is carried out by individual instructors, as in the Medical department.

The social and medical records of the patient are filed together; also a record of all patients who are refused admission, with notation as to why they were refused and the disposition of the case.

The influence on community health of an organization, such as the Curtis Clinic, would seem to me to have increasing advantage due to the methods of procedure which have been adopted. Dr. Samuel Bradbury, of the Pennsylvania Hospital Outpatient Department, in his report of 1930, has very aptly described three types of individuals in any community for which the outpatient department

does a great deal in the way of preventive medicine; that is, "the dispensing of health knowledge in the 'well-baby' clinic; the importance of early and cleanly care of minor surgical injuries, and the person who finds the stress of everyday life too much for him, presenting behavior or emotional difficulties, with the complaint of some organic pathological condition."

Another group of individuals whom the outpatient department would seem to help is well exemplified by the basis of our economic classification of patients: (*a*) Those individuals in the full-pay group judged by their income, size and responsibility of family and usual cost of medical service; and the occupation and required standard of living. This group, perhaps, offers the greatest difficulty in the admitting procedure because, oftentimes, they require further social investigation. We have tried the policy of investigating persons who attend the clinic well dressed, or perhaps are conveyed to the clinic by taxicab or even a private car, and often find such people are actually living up to the limit of their income, not allowing for the so-called "rainy day," which is exemplified by hospital bills, doctor's bills and the cost of medicine which may be necessary. The physicians in the Clinic very often complain of having to treat patients whom they feel are too well dressed to warrant treatment at dispensary rates, and very often complain of the fact that many of the patients who attend the clinics have private telephones in their homes. (*b*) The second group of patients are those who are unable to pay any clinic fees, for medicine or extra studies, such as *x*-rays and various laboratory tests. At the present time we have found this to be the predominating type of patient seeking dispensary care; and, unfortunately, many of the families have to be aided by the social service department, which functions in connection with each individual clinic.

In between the above stated groups we find the following economic classification: (1) Persons temporarily attending the dispensary because of unemployment, other financial emergencies or previous costly illnesses of themselves or family. This group varies greatly and does not make up a very large percentage of those in attendance; sooner or later they fall in the group who are able to pay a private physician. (2) Dispensary attendance necessitated because of some unusual condition with unusual expense of diagnosis or medical care required. This type of person is also usually a very temporary type, since they very often are referred either into the hospital

for more detailed studies or back to the referring physician after the diagnosis has been made.

Another phase of dispensary care represents, to my mind, a distinct economic advantage to any given community. The more expert available care the dispensary can give, the patient is saved long days of study and treatment in the wards of the hospital; ward beds are released for more acute conditions which formerly were occupied by patients undergoing diagnostic study. This service has economic advantage both to the patient and to the hospital, because many patients are able to continue their usual business activities, attend the Clinic during the lunch hour and keep up the wage income; and if an operation is deemed necessary, the preoperative studies can be carried out in the outpatient department prior to admission. Even in our short time of operation we have found that many patients have been able to be cared for quite satisfactorily in the Clinic who formerly occupied ward beds; this, of course, depends a great deal on whether or not the patient is a resident of Philadelphia and its suburbs. Jefferson Hospital has a great number of patients from out of town necessarily occupying ward beds, and this function of the Clinic in releasing more ward beds, has a very great influence on the immediate community in which we are located.

Whether or not the Curtis Clinic influences community health remains to be seen in the future; we are new and in the experimental stage, so to speak. Such clinics as the Prenatal Obstetrical clinic, the Well-Baby clinic and a newly organized clinic, the Medico-psychiatric clinic, certainly should aid in the medical education of the patients in our community. The stress laid upon preventive medicine, with increased facilities for instructing senior students in the College, should equip the men who will enter the practice of medicine in this community and other parts of the State with a new type of armament. Many of our students have expressed their gratitude that such an organization as the Curtis Clinic has been founded, so that they may learn to recognize disease in its early stages instead of the usual ward-class demonstration of more advanced pathology.

It is our aim to continue, in our small way, the philanthropy of the man whose munificent gift made possible the erection of the Curtis Clinic, Mr. Cyrus H. K. Curtis.

A REVIEW OF SOME RECENT MEDICAL LITERATURE RELATING TO MEDICAL ECONOMICS, COSTS OF ILLNESS, AND ORGANIZATION OF MEDICAL PRACTICE*

By WALTER S. CORNELL, M.D.

It is evidently impossible to discuss adequately the subjects of medical economics, costs of illness and organization of medical practice in the fifteen minutes alloted for the presentation of this paper. Possibly these subjects might be discussed, in the space of fifteen hours, sufficiently to bring the participants in the discussion to a level of information which would allow them to understand each other and to see at least dimly the various angles and merits of opposing arguments.

A reasonable knowledge of medical economics, costs of illness and organization of medical practice must presuppose a knowledge at least of (1) general sociology; (2) general economics; (3) the present status of the public health; (4) present costs of illness to the patient, his family and the public treasury; (5) the average income of practising physicians; (6) evenness of distribution of these medical incomes; (7) availability of medical service in rural and urban communities; (8) the professional knowledge of the most competent physicians and (9) of the least competent physicians; (10) reasonable equality of professional opportunity among physicians in the matter of hospital connections; (11) utilization of hospital laboratories for the widest benefit of the physicians of the neighborhood; (12) medical ethics in the matter of newspaper and radio publicity; (13) professional efficiency of physicians in private practice and those in salaried positions; (14) the influence of politics upon present day public health administration in states and cities; (15) the reasons for the existence of various healing cults; (16) group medical practice; (17) industrial medical practice; (18) the average income of nurses; (19) professional standards in nursing work; (20) group nursing; (21) use of second-grade nurses in the period of convales-

* Read January 29, 1932.

cence after the need of the more skilled registered nurse has ceased; (22) public health service which is confined in scope to prevention and diagnosis; (23) public health service which includes curative medical service, and finally (24) medical service which is identical with public health service because wholly socialized in character.

VOLUME OF RECENT LITERATURE. The *Bulletin* of the American Medical Association, official organ of the house of delegates, published nine times each year, in twenty-three of its last twenty-eight issues has published reports of official conferences, articles or editorials on the general subject of medical economics, sometimes related to the subject of medical ethics. Many of these articles are well worth reading. Others, particularly reports of remarks made by delegates, indicate strongly-formed opinions without evidence of study of the subject.

The *Index Medicus*, covering the period from January, 1931, to September, 1931, gives a list of sixty-nine articles on the subject of medical economics appearing in different medical journals, about 40 per cent of these appearing in the *Journal of the American Medical Association*. Between September, 1931, and January, 1932, the *Journal of the American Medical Association* published twelve additional articles or editorials.

The *Reader's Guide to Periodical Literature*, sold generally at bookstands, in its issues from July, 1930, to December, 1931, lists sixty-eight articles on the subjects of medical economics and costs of medical care which have appeared recently in various magazines. One of the sixty-eight publications mentioned (*Socialization of Medicine*, a digest by Edith M. Phelps, Vol. VII, Book 1, H. W. Wilson Co.) itself gives a list of 204 references and bibliographies.

The National Committee on the Costs of Medical Care (Dr. Ray Lyman Wilbur, chairman; Dr. C. E. A. Winslow, chairman of the executive committee) has published eleven major contributions and eight miscellaneous contributions to date, with recent announcement that the Chicago press is publishing fifteen additional studies. One pamphlet, *The Five-year Program of the Committee on the Costs of Medical Care*, gives the general plan of study and the personnel of the committee. *Miscellaneous Contribution No. 12* presents factual bibliography of studies made on the costs of medical care, with remarks concerning the contents of one hundred and fifty-one recent publications on that subject.

This scanty review of the literature on medical economics and

costs of medical care, most of it appearing during the past year, provides a list of 546 papers, reports and discussions.

Noteworthy Publications. The most noteworthy and most quoted utterance of any physician holding official position in the American Medical Association is that of Dr. Olin West, secretary of that great organization. . . . "The one outstanding problem before the medical profession today is that involved in the delivery of adequate scientific, medical service to all the people, rich and poor, at a cost which can reasonably be met by them in their respective stations in life."

A report on *Income from Medical Practice*, by Dr. R. G. Leland, director of medical economics, American Medical Association (*Jr. Am. Med. Assn.*, May 16, 1931), gives the incomes of the physicians of the United States as determined by a questionnaire mailed to 25,000 physicians (every fourth one in the *Directory of the American Medical Association*), and also presented to all of the physicians of the country by publication in four issues of the *Journal of the American Medical Association*, which is received by about 65,000 physicians. Approximately 2500 of the physicians circularized by mail, and approximately 4000 of the physicians who read the questionnaire in the *Journal*, took the trouble to answer it. The statistical summary indicated that, in the year 1931, the average gross income of the American Physician was $9664.40, the net income being about three-quarters of that amount. The report is very thorough in its classifications and analyses and well worth reading. The figures are considerably higher than those reported by the Committee on the Costs of Medical Care, in its investigation of the income of Philadelphia physicians—possible due in part to the fact that those physicians who answered the national questionnaire represented the more energetic and successful ones, and in part to the fact that Philadelphia, like other large cities, is overstocked with physicians.

The commission on medical education (1925), according to a statement recently noted, reports that 10 per cent of all physicians are specialists. This statement, while noteworthy, does not portray the true situation. In the rural districts and smaller towns practically all of the physicians are in general practice. Inspection of the American Medical Association *Directory*, edition of 1931, city of Philadelphia, two columns of names selected at random, shows that 59 physicians are in general practice (including five noted to be

hospital interns), while 33 are noted to be specialists. The Philadelphia situation is the basis for the remark that the family physician is disappearing from the scene.

The most comprehensive statement, in American literature, regarding the organization of medical practice in foreign countries is found in *International Studies: The Relation Between the Private and Official Practice of Medicine* (3 volumes), by Sir Arthur Newsholme, published for the Milbank Memorial Fund by The Williams and Wilkins Company, 1931.

Dr. C. E. A. Winslow, professor of public health, Yale University, and chairman of the executive committee, National Committee on the Costs of Medical Care, has recently published an interesting article in the *Yale Review*. He calls attention to the fact that while communicable diseases have been fought successfully (due largely to sanitary engineering, better hygienic living standards, and comprehensive immunization measures), there has been an increase in the incidence of heart disease, kidney disease and cancer, and possibly an increase in nervous and mental diseases. There is a woeful lack of support of community health programs, and failure on the part of the public to seek and demand preventive service from the private physician. The public uses great quantities of drugs and nostrums, and services of medical quacks, and tends more and more to seek charitable medical service. Physicians are tending more and more to settle in cities, so that there is an oversupply of them there and an undersupply in the rural districts, and moreover many of the rural physicians are over fifty years of age—soon to discontinue their active work. The average physician, because of high expenses and low collections, can look forward to a net income of less than $3500 per year. A recent survey by the U. S. Public Health Service in Hagerstown, Maryland, revealed that 54 per cent of all the illness occurring received no medical care. Similar studies by the Metropolitan Life Insurance Company gave a figure of 25 per cent. In many cases, the medical care given could not have been first class, as it was given without possession by the attending physician of *X*-ray or laboratory facilities for exact diagnosis.

Dr. Winslow states that the average expense for medical care is about $25.00 per year per person, but while this is not high, the average working man has uses for all of his income, does not budget his expenses, and is not prepared to meet the costs of medical service because the latter do not come at an even annual rate, but often

are met in the form of bills for a great deal of medical service, due to serious illness in the family after years of fortunate freedom therefrom. The worker with a large family is especially unprepared to pay anything more than a few dollars at any one time. He gives statistical figures supporting his statement, gathered by the investigators of the National Committee on the Costs of Medical Care.

He notes that in New York City one-half of all the hospital beds are free beds (possibly to illustrate the unpreparedness of the public to pay for hospital service because of lack of planning to pay for such service), and cites the results of a study of 1788 families made by the Committee on the Costs of Medical Care. Forty per cent of these families spent less than $25.00 for medical care during the year of the investigation, but twenty families were called upon to spend $500.00, and three families had to spend $1000.00 or more and this was more than one-half of the family annual income.

Hence Dr. Winslow advocates some form of health insurance to be paid by the worker, possibly with some public subsidy to make the amount sufficient to provide satisfactory medical service with satisfactory remuneration of the physicians. He emphasizes that any such plan should be governed by professional personnel and that the fullest possible personal relationship should be maintained between the physician and the patient.

Mr. Evans Clark (*Atlantic Monthly*, September, 1930) states that the average man of the middle class carries insurance on his house and insurance on his automobile, but does not carry insurance against the contingency most likely to occur, namely, sickness in himself or his family. (Possibly this is so because the individual, if out of funds, can always obtain free medical service, but cannot obtain a free house or free automobile.—W.S.C.). Mr. Clark believes that a successful unified health service must include three essentials: (1) periodic health examinations, (2) group practice, as exemplified by the Mayo Brothers' Clinic, and by about sixty other group clinics averaging ten to twenty physicians per group, (3) an insurance principle by which the financial risks due to illness are spread evenly throughout the years, fixed and regularly paid.

Miss Constance Kent, after reviewing the publications of the Committee on the Costs of Medical Care, states that the thirty-six million wage-earners of the United States lose at least two hundred and fifty million work days per year. It is estimated that illness entails decrease in earning capacity of about two billion dollars,

and that deaths which might have been prevented represent additional annual decrease in earning power of about six billion dollars. Mr. Michael M. Davis, Director of Medical Service, Julius Rosenwald Fund, is quoted with the statement that the direct cost of illness is slightly under three billion dollars per annum. Of this amount, hospitals and clinics are reported to receive 30 per cent, purveyors of drugs, medicines and appliances, 25 per cent, private physicians 25 per cent, dentists and nurses, 13 per cent, and practical nurses, midwives, nonmedical practitioners and quacks, 6 per cent. The remaining 1 per cent represents the amount spent on preventive work.

Miss Kent's article may be taken as representative of the viewpoint of many economists and social workers. She remarks that formerly patients were afraid to go to hospitals for fear they would die there, but now they are afraid to go for fear they will not come out solvent, and that there is a crescendo accusation against medical charges in general, due to special costs of *x*-ray, laboratory, specialists' and hospital service.

The reports on health service in the public schools show very clearly the great numbers of schoolchildren seeking free or near-free treatment when suffering from eyestrain, adenoids, diseased tonsils and dental caries. The annual report of the Division of Medical Inspection of Public Schools, Philadelphia, for the year ending June 30, 1931, shows that 19,554 cases of eyestrain were recommended for treatment (procurement of needed glasses). Of these 6472 were treated and 13,082 were not treated. Conditions were not quite as bad as these figures at first indicate, because the 13,082 cases "Not Treated" included many pupils whose parents had repeatedly refused to seek medical care, for many years, but they are nevertheless a commentary on the neglected condition of this large group of children. The small number and proportion of the pupils seeking medical service from private physicians (ophthalmologists) is remarkable—only 1850. On the other hand, 3686 secured refraction of the eyes at hospital clinics. There were 936 pupils who secured treatment from "Other Sources," mostly optometrists, with a few treated by osteopaths and chiropractors!

The Philadelphia school medical inspection figures for tonsils and adenoids, in the report just noted, paralleled those for eyestrain. Of 3062 cases treated by operation, only 412 were treated by private physicians, whereas 2650 were treated at hospitals as cases originat-

ing in the outpatient clinics. While it is good practice to perform these operations at hospitals rather than at home, in order to have the patients under favorable surroundings in case of postoperative hemorrhage, it is believed that whatever charges were made by the hospitals (usually ten to fifteen dollars for the use of the operating room and one night in a ward) were retained entirely by the hospitals, the surgeons receiving nothing.

COMMENT. The literature which has been reviewed indicates a wide dissimilarity in viewpoint. Generally speaking, the economists, sociologists, physicians engaged in public health work and social workers have a clearer conception of the unsatisfactory health situation existing than do the physicians who are in private practice. This is natural since the private physician, as a rule, concentrates his attention on the sick who come to him for treatment, and the medical journals intended primarily for physicians in private practice devote their space to diagnosis and treatment, rather than to comparative death rates and sickness rates, costs of illness and hygiene.

On the other hand, those having the social medical viewpoint seem to forget that much of the ill health existing is due to poverty and ignorance—the same basic condition that breeds immorality and crime. It would appear that the fundamental procedure to assure health, happiness and morality is not a direct attack on any prevailing system of medical practice, but rather a first attack on the existing social order, in which a large proportion of the public have not the means to purchase anything beyond the bare necessities of life.

On the other hand, also, the proposals made to socialize or semi-socialize the practice of medicine, in this country, are unsatisfactory because they are indefinite in the matter of protection of the physician, and the system of medical care, from the baneful influence of local politics—destructive to professional morale wherever encountered. It is doubtful if economists, sociologists and college professors who regard this point lightly in the case of the medical profession would view with equanimity any attempt to place them, themselves, under local governmental control. Life would be quite different.

The first step in the demonstration that medical practice, under state or municipal control, may be freed of political influence, is to demonstrate it by divorcing health departments from political influence. With every change in political administration, a new

health officer appears. First-class physicians, other than those with a missionary spirit, hesitate to enter the field of public health, with proper preparation by postgraduate study, because of its uncertain tenure.

Starting with a positive pronouncement that the basis of medical care should be the system of private practice, there should be a generous recognition, that where public health measures or industrial hygiene measures produce much better results, they should be employed. The history of communicable disease control is a recital of the efficiency of public health measures in this field. Immunization against smallpox and against diphtheria, the eradication of hookworm by comprehensive clinical measures plus better sanitation, the eradication of malaria and of typhoid fever by sanitary engineering measures, are illustrative examples. In view of the recent prominence given to the "Detroit plan" of diphtheria immunization, it is proper to note that the results, measured in lives saved and reduced costs of illness, are far better in Philadelphia where the Health Department has used its own comprehensive measures with coincident encouragement of private physicians to immunize as many children as possible with use of immunizing serum furnished free of charge. It is far better for the medical profession to recognize its own worth in the field of industrial medicine and public health than to engage in internal controversies where the better plan is evident to the average intelligent lay person. The medical profession rather should unite to conserve the health of the community by offering its own services on the best basis, thus discouraging resort to cultists and outright quacks.

The two principal criticisms of the existing system of medical practice are: (1) the multiplicity of required medical service in a given case of ill health, due to the increasing number of specialties and the tendency of general practitioners, especially in our large cities, to refer cases to specialists rather than to undertake their treatment; (2) the high cost of specialists' services. The remedy to the first of these situations, which undoubtedly exists in some degree, is the rehabilitation of the general practitioner. The slogan of the medical profession, in the present situation of controversy, is "consult your family physician." The family physician will be able, in professional knowledge and resources, to care for his patient satisfactorily proportionately as the diagnostic facilities of the large hospitals are made available to him, and the semiprivate rooms

and private rooms are at his disposal for the care of his patients. Regarding low-scale fees by specialists, when treating the poor, the difficulty now existing is not any unwillingness on the part of most specialists to make the fee suit the patient's purse, but rather a lack of realization or confidence on the part of patients that such is the case. There is a hesitancy on the part of both specialists and patients to discuss the general amount of the fee before the service is rendered. Much of the complaint by ophthalmologists and otolaryngologists that people able to pay a small fee are seeking treatment at hospital clinics would be met by a definite statement by these specialists that they will be glad to treat the poor, and those of limited means, on the basis of their capacity to pay. A definite "reduced-fee" schedule might be set up by the county medical societies, graded according to family finances.

It would be well for physicians in private practice to use better business methods. It is impossible to give consideration to financial welfare without commenting on their hesitancy, born of delicacy, timidity, or vague ideas of dignity, to expect cash payment when dealing with strangers whose credit is not established. Millions of dollars are lost annually in unpaid fees. A person who engages and occupies a hotel room, without the money to pay for that accommodation, is guilty of fraud. The medical profession might unite to secure similar legislation protecting the physician.

The plan of health insurance, free of commercial or political control, has much to commend it from the standpoint of increased medical income. Certainly, as long as people do not save and have no money, the doctor cannot be paid—unless the state pays him, and that alternative is opposed by the majority of the medical profession.

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AN EXPERIMENT IN PSYCHOANALYSIS*

By EARL D. BOND, M.D.

The object of this paper is to present the considerations which surround the introduction of analysis into a medical community. Of psychoanalysis as such I am unqualified to speak, but it seems fair to leave my hearers in no doubt as to what I mean by the term. The definition that I have accepted is that psychoanalysis is a study of the dynamics of mental life by a special technique developed by Dr. Freud. The possible relationships of analysis, so defined, to medicine are so important and involved that there is reason for physicians and surgeons as well as for neurologists and psychiatrists to know more about them. At the least it concerns physicians because their patients are learning about it from the stage, from the radio and from popular magazines and books.

In Philadelphia, particularly, discussions of analysis have tended to be nonmedical. Social workers have gone to Europe or New York and returned to put into practice analytic principles. Medical Philadelphia has entered not at all into that new philosophy of social service which, outside of this city, is labeled Philadelphian. Some advantages arise from this situation. The medical profession is not responsible for these new procedures; it can reserve judgment, not only on social questions but on psychoanalysis itself; in contrast to other cities, physicians here have not been forced to take sides too hurriedly.

But the advantages of ignorance do not last forever. Psychiatrists especially are kept in an uncomfortable position if they cling to them too long. They are pressed for advice about this new therapy by patients who admittedly get no help from present Philadelphia resources. They have seen patients turning to other cities for analytic help and bringing back experiences which are difficult to evaluate. In all psychiatric societies psychoanalysis now has a recognized place on the program.

Under this pressure psychiatrists grouped about the Institute of

* Read March 2, 1932.

the Pennsylvania Hospital decided they could no longer keep from taking a firsthand view of this new development. In this decision they were preceded by their colleagues in the Massachusetts General and the New York Hospitals. Such a view can well be taken by a group whose individual differences in character and in education and in nearness to the experiment can be used to check individual prejudices and to add importance to conclusions.

A first step was to secure an expert who stood not on the fringes but in the center of the psychoanalytic movement. It was important to get a well-trained physician with psychiatric experience. Three psychoanalysts and three psychiatrists without analytic leanings, all of whom had a knowledge of the international field, were asked to suggest this expert and all of them recommended Dr. Herman Nunberg, of Vienna, who had had general psychiatric training under Professor Bleuler and analytic training under Professor Freud. Dr. Nunberg was secured for the university year of 1931–1932. A second step was to refer to him patients, among them four Institute psychiatrists and one internist. The analyses of these physicians is the nucleus of the experiment and its success or failure will be decided here. But the results of these analyses from the personal and from the scientific angles will naturally not be known until their completion some time in the future. The antecedents of these physicians were very well known before the experiment began. In looking at their medical antecedents it is interesting to note that these five men interned at the Jefferson, University, Philadelphia General, Montefiore and Massachusetts General Hospitals. It is of importance that the senior psychiatrists are not being analyzed, but are interested as a control group and as critics.

A third step was the establishing of lecture and conference courses under Dr. Nunberg for a wider group of physicians.

These are the three steps which have been taken and which cannot now be reported upon. Their value will be determined later.

But there are other matters not quite in line—side issues—which can be now commented upon briefly. There is growing at the Institute a record of the experiences of its patients and of friends of the physicians with analyses elsewhere. Knowing of our interest people have come to us sometimes simply with a history of a past experience and sometimes with a question about a present

need. In some of these cases apparently there has been a complete relief of nervous symptoms. And in some cases there has been no relief. The individual variations are endless, from the simple account of relief from a twenty-year-old gastric distress after eight months' treatment to a story of a search lasting eight years without any relief from the best analysts. It is fair to state that included in these reports that drift in casually are some which record still longer searches among neurologists and psychiatrists of what might be called the regular school, with no better success. In fact in all of these patients there is evidence of a desperate seeking for health which will turn, if disappointed in local physicians, to analysts reputable or disreputable elsewhere, to Christian Scientists, to chiropractors and the rest.

A sidelight upon the matter has come from a small group of patients who could properly be taken for analysis by those of the Institute physicians who had had some training before the beginning of this experiment.

One of these cases showed in infancy impulses to be cruel to animals which the patient had long forgotten and, very clearly as a reaction to these impulses, an adult life unselfishly devoted to the relief of suffering.

Another sidelight comes from a number of children under ten years of age, residents of a school at the Institute. Analytic technique claims to get back to childish thinking. A special technique, set hours of companionship and play with a qualified older person allow us to get at the thinking of these children of four to eight years of age. The comparisons are fascinating and their scientific value cannot be doubted. One ought by means of these comparisons to know whether or not adults thinking backward have gotten at genuine childish thinking or only a thinking disturbed by all the experiences which have come in between them and the later recollecting of them. For instance, one seven-year-old boy says directly:

"See what I am doing? I am Mickey Mouse and when I go up in an airplane and then I come down fast and shoot somebody. I shoot Mr. Bad Man because he tried to kill my little boy; but he did not kill him, he just hurt him and now I killed HIM!"

However, I turn to a different and most important set of patients, those suffering from psychoses in the Department for Mental Diseases. Here under the care of physicians who have not been

analyzed or not in any way committed to its theories, but who are engaged in the lectures and conferences, are people who are, so to speak, Nature's experiments in analysis—people who have by disease been stripped of their "esthetic and moral restrictions and inhibitions." So that they express in word and action a mental content which is hidden from the normal person except in the artificial procedure of analysis. From such people, if anywhere, should come unbiased criticism. It is of importance that this year the physicians who have been making these observations have been, in common with psychiatrists in many other hospitals, obtaining remarkable changes for the better by new drug therapies. So that they also are in the condition of not being biased.

There are a dozen cases where one of the outstanding characteristics of the personality before any psychosis began was an unusual fussiness about and rejection of food. And in most of these food faddists are also such childish things as fussiness about dress, temper tantrums, girl shyness. Here follows a collection of mouth habits with thoughts about the mouth as expressed by grown-up patients:

The first man had a neurasthenic mother who never gave him proper food. He became a brave young man who was doing well at the Naval Academy until he began to devote all of his time to nail-biting, the study of his food and of his clothes.

A second young man lives on salads and chicken only; he slept with his mother "to protect her."

A third man lived on salads and saltine crackers and scrubbed his teeth until his gums were sore.

A fourth man was discovered shooting air through his lips "to make himself cleaner."

A woman swallowed her wedding ring.

And here are the remarks of two young men: "I am going to cut my mother up into little pieces and eat her." "I produce and create food by thought processes."

One of the men and one of the women seem to have had their lives centered about their gastro-intestinal tracts for forty-eight and thirty-six years, respectively. The woman, sickly and pampered all her life, never without a digestive disturbance as a child, wanted to be a missionary but was prevented by typhoid fever, of all diseases; now she has nausea which interferes with all her plans. The man, "morally perfect," has never been free from worry about a chronic diarrhea and anal distress.

I quote here the thoughts of a patient occupied only with her own inner needs, undisturbed by having many persons or no one in the room, by having the door open or shut, unable even to recognize those about her. She says: "Here is the Manship—in the submarine. I took a big jump

back to childhood—my mother must have suffered as I did—up in some high place and water all around in the bottom of the ship—if I had to live over my mother's life—and find out everything—I want to go back and be young—I probably was the real twin and so I died."

Now there are obvious bases for comparisons here. Patients being analyzed are trying to remember and report their childish ways of thinking; children, aged from four to ten years, are reporting their thoughts directly; psychotic patients are from one point of view giving up their struggle with reality to go back to a childish happier time when fantasy was used without regard for facts.

And here is a discussion on the father-daughter relationship by an adult under analysis, by a child and by a psychotic patient:

The first patient, a girl, aged twenty-one years, had gone to an internist about the return of fears which had prostrated her at the age of sixteen years. The internist, having neither the technique nor the time to enter further into the case, referred her for analysis. The diagnostic results of some months' work can be summed, grossly, as follows:

An infantile autoerotic attachment to her father stands out, which is desperately objected to by a part of her mind which has associated a fear of death with autoeroticism. Fantasies and dreams show that different men—friends, brother, lover—are finally only representatives of her father. For instance, in a dream the person approaching was first a man, then her brother, and then her father. She said: "I can tell you these dreams, because they mean nothing to me."

A girl, aged seven years, in the school, on coming into the playroom, said to the physician: "You be the father and I'll be the mother!" The next day she said: "We'll have to have a father and mother," and then proceeded to call the physician by her father's first name, while she gave her mother's first name to herself. The third day the physician was to be father and she would be the wife. She said: "My father likes me better than my sister." A little later—"My sister is dead—not really."

And a last example without camouflage: A single woman, dean of a woman's college, in her involutional melancholia, said: "I am large here in my abdomen. I am what you call pregnant—by my father. I have had sexual relationships with myself and my father, and generations yet unborn are to suffer."

Comparisons like this are being multiplied. More than can be shown in illustrations, the situations which call forth the patient's remarks and behavior are to be seen living and developing and dynamic under the physician's eyes, and I cannot help thinking that we shall learn from such experience.

This, then, is the setting of an experiment in psychoanalysis—a setting which offers unusual chances not only to check false conclu-

sions but to confirm valid ones. It places analytic research alongside chemical and neurological research, with which there is no conflict whatever.

If the assumptions of psychoanalysis are, by careful work in the future, shown to be well grounded, it is already obvious that some of the fundamentals of psychiatry and of all medicine will be changed.

DISCUSSION

Dr. Edward A. Strecker: When Dr. Bond said that "heretofore discussions about psychoanalysis in Philadelphia had been on a non-medical basis" he made what might be called a very minimum statement. He might have used such adjectives as "heated," "superheated," "violent" and still not have done justice to the type of discussions that have prevailed even within the sacred precincts of this College. It was interesting to hear his calm, dispassionate statement concerning psychoanalysis.

Psychiatrists should be quite willing to acknowledge the debt that psychiatry owes to psychoanalysis. When I began to study psychiatry, we had as our guide that master mind, Emil Kraepelin. He had written a remarkable textbook and it was the bible of psychiatric students. There was page after page of careful, conscientious descriptive observation of the symptoms Kraepelin saw when he examined his patients. So did we study out patients. Whatever we saw, hallucinations, delusions, etc., we simply wrote a careful and accurate description, being very little concerned or unduly curious about what might underlie this or that particular symptom or sign. If a patient who had catatonic dementia precox would lie for a long time on the floor with arms outstretched in the form of a cross, we simply reported it as an interesting clinical phenomenon. This was very much like telling a student of physical diagnosis to apply a stethoscope over a certain area of the chest and asking him to remember that the peculiar sound he hears means aortic regurgitation. So the student makes auditory remembrance of that particular sound, and when he hears it again he thinks of aortic regurgitation. This would scarcely be satisfactory. The student ought to know something about the underlying cardiac condition and mechanics. Psychoanalysis attempted to explain the mechanics of neurotic and mental symptoms. It did venture to make guesses—scientific guesses—as to the real reasons, for instance, as to why a patient should lie outstreched upon the floor in the shape of a cross; or why another patient walking along the street should believe that every passer-by was indicating in some way a belief that the patient was a sex pervert. Psychoanalysis tried to uncover basic psychopathology. Furthermore, it encouraged the patient to talk and it encouraged psychiatrists to listen to what the patient had to say. Probably, for the first time, the patient was given an opportunity to talk himself out and every psychiatrist knows the very great value of confession or mental unburdening. So far so good.

To evaluate psychoanalysis as a system of therapy is something else. I think psychoanalysts will admit many defects. It cannot be evaluated (and psychoanalysts will object to this) because it is not capable of scientific proof. This does not mean that it is not valuable, since many things that cannot be scientifically demonstrated are immensely valuable. It is a cumbersome and lengthy and expensive proceedure. It is difficult to determine when a suitable stopping point has been reached. Personally, I feel (psychoanalysts may rightly take exception) that suggestion is not excluded. I doubt whether the intimate relationship between patient and physician can exist without considerable suggestion passing from the doctor to the patient. Even though the psychoanalyst is presumed to be merely a receiving apparatus, I still think it is beyond his power not to suggest to the patient and thereby influence the patient somewhat by his own attitude.

Finally, the value of the psychoanalytic experiment at the Institute is obvious. Psychoanalysis is an important method of psychotherapy. It is utilized wherever psychiatry is practised. It will survive in some form, perhaps not its present one, and become solidly incorporated into the body of psychotherapy. Let us find out all we can about it.

THE INCREASING INCIDENCE AND MORBIDITY OF APPENDICITIS*

By JOHN O. BOWER, M.D.

The first meeting of the Association of American Physicians was held in Washington, June 17, 1886. It was called to order soon after 10 o'clock in the reading-room of the Army Medical Museum by Dr. Francis Delafield. Dr. James Tyson was temporary chairman. Fifty-eight physicians were present, 14 from New York, 13 from Philadelphia, 7 from Boston, 6 from Baltimore, 2 from Montreal; the remainder from various cities of the United States.

On the afternoon of June 18 Dr. Fitz read his memorable paper on "The Diseases of the Appendix Cæci." He had collected 257 cases of perforating inflammation of the appendix; all had peritonitis. In 1890 the reported death-rate of appendicitis was 26 per cent; practically all of these cases had peritonitis when admitted to the hospital. In Philadelphia, in 1930, of the 3095 patients admitted to 28 hospitals, 35.4 per cent had peritonitis, yet diagnosis was made in less than 5 per cent of the cases. During this same year the mortality of spreading peritonitis was 26.27 per cent. Dr. Fitz, at this first meeting, spoke about the chief cause of the high mortality of spreading peritonitis. "A cathartic or a laxative may be demanded by the patient or friends and an enema be thought desirable as a diagnostic aid. It is to be remembered that these may be the means of at once exciting a general peritonitis."

Forty-six years have elapsed since Dr. Fitz made this statement. During this period the management of acute appendicitis has improved, but the mortality of spreading peritonitis is about as great as then, chiefly because we have permitted the population at large to remain in ignorance regarding delay in hospitalization and the use of laxatives. Whether these two factors alone are responsible for the increasingly high death-rate in the United States is

* Read March 2, 1932.

impossible to state, but we do know that the death-rate in this country is 59.3 per cent higher than the city of Mexico, 70 per cent higher than Germany, 70 per cent higher than Scotland, 98.7 per cent higher than New Zealand, 109.5 per cent higher than England and Wales and 131.8 per cent higher than The Irish Free State, and 313.5 per cent higher than Italy. It may be, as Morris states, in Italy the surgical work in appendicitis is in the hands of master surgeons or that the out-of-doors occupation in agricultural pursuits gives a higher degree of oxidation of toxins.

The responsibility for the management of patients suffering with appendicitis is the surgeon's, yet factors beyond his control are mainly responsible for the mortality. The average surgeon can successfully manage patients with appendicitis and spreading peritonitis who have not been given laxatives, but a laxative-induced spreading peritonitis is in the majority of instances fatal. In Philadelphia, from 1928 to 1931, inclusive, 593 patients died from appendicitis: 314 gave a definite history regarding laxatives; 294, or 93.63 per cent, had received a laxative; 238 of the 294 died because of a spreading peritonitis.

The mortality from typhoid fever and tuberculosis has been tremendously reduced because of prophylactic medicine. Surgeons should universally advocate prophylactic surgery, as it pertains to spreading peritonitis. Informing the public of the dangers of delay and the administration of laxatives would reduce the mortality of appendicitis 50 per cent in ten years, and in twenty years patients admitted to hospitals with spreading peritonitis would be as rare as patients suffering with typhoid fever are today.

During the past three years such a campaign to reduce the mortality of acute appendicitis has been conducted in Philadelphia. The Department of Public Health has surveyed 28 hospitals from 1928 to 1931 inclusive. With the coöperation of the Philadelphia County Medical Society and the Philadelphia Association of Retail Druggists an effort has been made to inform the public, through the family physician, of the dangers of delay and the administration of laxatives. This campaign has met with some success: In Philadelphia, from 1909 to 1919, the average yearly mortality-rate for acute appendicitis was 12.5 per 100,000 population. From 1919 to 1929 the rate had risen to 15.5 per 100,000, an increase of 24 per cent. During the past three years, the average has been 14.17 per

100,000. In 1931 the mortality-rate was 13.9, the lowest in the last ten years.

Table 1 shows that of the patients admitted to the 28 hospitals of Philadelphia in the past four years with an acute catarrhal appendicitis only 1 in 204 died; if they had an associated local peritonitis 1 in 35 died; but if they were unfortunate enough to have a spreading peritonitis then 1 in 4 died.

TABLE 1.—APPENDICITIS—MORTALITY (1928 TO 1931 INCLUSIVE)

	Cases.	
Acute appendicitis	6,952	34—1 in 204 died
Acute appendicitis with local peritonitis	2,745	78—1 in 35 died
Acute appendicitis with spreading peritonitis	1,661	481—1 in 4 died
	11,358	593—1 in 19 died

During these four years 11,358 patients were operated upon: 10,765 lived, the average time between onset of symptoms and operation being 54.3 hours; 593 died, the average time between onset of symptoms and operation being 84.39 hours.

TABLE 2.—MORTALITY—DELAY

	Total. cases	Total deaths.	Per cent mortality.	Average time, onset symptoms to operation. Recoveries, hours.	Deaths, hours.
1928–1929	5,121	306	5.97	56.50	95.06
1930	3,095	149	4.81	48.67	68.05
1931	3,142	138	4.39	52.55	78.38
	11,358	593	5.22	54.30	84.39

Table 3 shows the increase in mortality with the increase of time between onset of symptoms and operation.

TABLE 3.—MORTALITY—DELAY (1928 TO 1931 INCLUSIVE)

Total cases admitted to hospital.	
3,589	Within 24 hours— 78 or 1 in 46 died
4,107	Within 48 hours—199 or 1 in 21 died
1,558	Within 72 hours—113 or 1 in 14 died
2,104	After 72 hours —203 or 1 in 10 died
11,358	593 or 1 in 19 died

Table 4 shows the chances of recovery of those who had and those who had not received a laxative.

TABLE 4.—MORTALITY—LAXATIVES (1928 TO 1931 INCLUSIVE)

Cases.	
1,185	Not given laxatives —19—1 in 62 died
3,412	Given 1 laxative —204—1 in 17 died
378	Given more than 1 — 59—1 in 7 died
6,383	No definite history —311—1 in 21 died
11,358	

Table 5 shows the death-rate in each group of those who took 1 laxative.

TABLE 5.—MORTALITY—LAXATIVES (1 LAXATIVE ADMINISTERED)

	Total cases.	Given 1 laxative.	Deaths.
1928–1929	5,121	1508	85—1 in 18 died
1930	3,095	889	62—1 in 14 died
1931	3,142	1015	57—1 in 18 died
	11,358	3412	204—1 in 17 died

Table 6 shows the uniformly high death-rate of those who received more than 1 laxative.

TABLE 6.—MORTALITY—LAXATIVES (MORE THAN 1 LAXATIVE ADMINISTERED)

	Total cases.	Given laxatives.	Deaths.
1928–1929	5,121	163	32—1 in 5 died
1930	3,095	103	15—1 in 7 died
1931	3,142	112	12—1 in 9 died
	11,358	378	59—1 in 7 died

Spreading peritonitis is the cause of 81 per cent of the deaths from appendicitis, where a definite history of laxative administration was obtained from the clinical records of those who died over 90 per cent had received a laxative.

TABLE 7.—MORTALITY—LAXATIVES SPREADING PERITONITIS

Year.	Deaths.	Definite history laxatives.	Laxative positive.	Per cent.
1930	149	83	72	86.74
1931	138	71	69	97.18
	287	154	141	91.56

Of those not given laxatives 1 in 62 died; those given 1 laxative 1 in 17 died; more than 1, 1 in 7 died.

Table 8 shows the kinds of laxatives taken, with results.

TABLE 8.—KIND OF LAXATIVES

	1930.			1931.		
	Recoveries.	Deaths.	Total.	Recoveries.	Deaths	Total.
Citrate	168	10	178	170	12	182
Castor oil	140	21	161	167	7	174
Salts	93	10	103	168	4	172
Milk of magnesia	49	3	52	72	4	76
Ex-lax	38	0	38	55	1	56
Mineral oil	14	0	14	14	0	14
Cascara	13	0	13	15	1	16
Sal hepatica	5	3	8	11	0	11
Alophen	8	0	8	9	0	9
Seidlitz	8	0	8	7	0	7
Calomel	7	0	7	1	0	1
Pluto	7	0	7	7	0	7
Feenamint	5	0	5	6	1	7
Syrup of figs	4	0	4	4	0	4
Castoria	3	0	3	5	1	6
Phenolax	2	0	2	1	0	1
Phosphate of soda	2	0	2	0	0	0
Soda bicarbonate	0	0	0	4	0	4
Psylla seed	1	0	1	0	0	0
Senna	0	0	0	2	0	2
Agarol	0	0	0	2	0	2
Rhubarb	0	0	0	1	1	2
Sehenek's pills	0	0	0	1	0	1
Petrolagar	0	0	0	1	0	1
Multiple laxatives	88	15	103	100	12	112
Kind not mentioned	260	15	275	235	25	260
Had no laxative	397	5	402	388	2	390
No history	1634	67	1701	1558	67	1625
	2946	149	3095	3004	138	3142

Tables 9 and 10 show the comparative mortality of acute appendicitis in cities in the United States with a population of 300,000 or over.

TABLE 9.—1930 DEATH-RATE FROM APPENDICITIS IN 25 CITIES OF OVER 300,000 POPULATION

	Population.	Deaths.	Death-rate per 100,000.
Philadelphia, Pa.	1,953,423	282	14.4
Portland, Ore.	303,606	44	14.5
Seattle, Wash.	366,552	54	14.7
San Francisco, Calif.	636,866	96	15.1
Indianapolis, Ind.	365,130	56	15.3
Los Angeles, Calif.	1,250,857	191	15.3
Jersey City, N. J.	317,254	50	15.8
Rochester, N. Y.	328,762	52	15.8
New York City, N. Y.	6,991,957	1110	15.9
Pittsburgh, Pa.	701,974	115	16.4
Cleveland, Ohio	902,450	155	17.2
Baltimore, Md.	806,297	147	18.2
Chicago, Ill.	3,389,508	618	18.2
Detroit, Mich.	1,576,124	295	18.7
Louisville, Ky.	308,843	58	18.8
Buffalo, N. Y.	575,088	112	19.5
Washington, D. C.	487,824	98	20.1
Milwaukee, Wis.	580,596	119	20.5
St. Louis, Mo.	822,909	175	21.3
Boston, Mass.	781,828	168	21.5
Newark, N. J.	442,874	100	22.6
New Orleans, La.	460,152	106	23.0
Minneapolis, Minn.	465,979	108	23.2
Cincinnati, Ohio	452,130	109	24.1
Kansas City, Mo.	401,207	106	26.4
	25,670,190	4524	17.6

TABLE 10.—1931 DEATH-RATE FROM APPENDICITIS IN 26 CITIES OF OVER 300,000 POPULATION

	Population.	Deaths.	Death-rate per 100,000.
Houston, Texas	310,997	23	7.4
Indianapolis, Ind.	370,206	49	13.2
Philadelphia, Pa.	1,966,351	274	13.9
Baltimore, Md.	813,469	118	14.5
Los Angeles, Calif.	1,318,058	193	14.6
Portland, Ore.	306,780	47	15.3
San Francisco, Calif.	649,844	101	15.5
Rochester, N. Y.	332,047	53	16.0
Jersey City, N. J.	318,965	51	16.0
New York City, N. Y.	7,088,966	1157	16.3
Pittsburgh, Pa.	679,672	112	16.5
Milwaukee, Wis.	592,904	101	17.0
Seattle, Wash.	371,658	64	17.2
Chicago, Ill.	3,458,053	612	17.7
Louisville, Ky.	316,565	56	17.7
Detroit, Mich.	1,638,217	291	17.8
Cleveland, Ohio	912,954	165	18.1
Washington, D. C.	492,539	93	18.9
St. Louis, Mo.	827,900	164	19.8
Newark, N. J.	445,697	92	20.6
Buffalo, N. Y.	581,101	120	20.7
Minneapolis, Minn.	474,496	103	21.7
Boston, Mass.	785,193	178	22.7
New Orleans, La.	467,417	117	25.0
Cincinnati, Ohio	457,205	129	28.2
Kansas City, Mo.	408,866	117	28.6
	26,386,120	4580	17.4

We believe that the low mortality in Philadelphia is due for the most part to the publicity campaign inaugurated in 1929. The following is an epitome of the plan:

AN EPITOME OF THE PLAN FOR THE REDUCTION OF ACUTE APPENDICITIS IN PHILADELPHIA AS IT PERTAINS TO THE INDIVIDUAL HOSPITAL. Any plan to combat successfully the increasing mortality of acute appendicitis must include a publicity program.

The basic thought underlying the campaign is to teach the public the dangers of delay and the abuse of laxatives. Publicity must deal with facts. These facts are obtained by an analysis of the clinical records of the hospitals and their value is determined by the care with which the abstracting is done and the accuracy of the analysis. The abstracting can be done by a member of the record room personnel, but one of the staff must inspect the work and analyze the abstracts. The man selected for this should not only be interested, but he should be interested in clinical research, he must be willing to supervise the details incident to statistical

studies. He should be selected by the chief of staff and the medical director of the hospital and should be chosen without regard for rank or individual preference.

The Plan

1. An analysis of all clinical records of acute appendicitis, both private and ward, of the previous or better, the two previous years should be made. This should include name, date, age, operator, time in hospital in days, time between onset of symptoms and operation in hours, definite history of laxatives, recurrent attacks, temperature and pulse, nausea and vomiting, anesthesia, operation, drain, leukocyte count, duration of operation, pathology and remarks.

2. The results of the analysis with the plan for the campaign should then be presented to the medical staff and those physicians sending patients to the hospital. Hospital and not individual mortality should be discussed frankly, with special reference to spreading peritonitis which is responsible for over 90 per cent of the deaths.

3. A letter should be sent to members of the staff and outside physicians referring patients to the hospital. It should contain:

(*a*) A summary of the analysis of clinical records, number of cases, mortality, percentage admitted to hospital with peritonitis, average time between onset of symptoms and operation of those who lived and those who died, and the percentage given laxatives.

(*b*) A request for coöperation in affixing the enclosed stickers to their monthly statements to patients. The sticker reads as follows:

WARNING

In the presence of abdominal pain

Never *give a laxative*
Give nothing by mouth
Apply ice-cap or hot-water bottle
Call your family physician

Abdominal pain which lasts over six hours is usually serious

This warning is published by the Philadelphia County Medical Society and endorsed by the Department of Public Health

(*c*) At varying intervals, three to six months, an analysis of clinical records should be made and letters noting any progress, together with stickers should again be sent to staff and outside physcians.

(*d*) At the end of the current year a complete survey should be made and the results reported at a meeting as above, followed by letters containing statistics showing progress.

(*e*) The survey and campaign should be conducted annually.

INCIDENCE OF TUBERCULOSIS IN INFANTS AND PRE-SCHOOL CHILDREN AND THE CONDITIONS UNDER WHICH THEY BECOME INFECTED*

By J. C. GITTINGS, M.D.

(From the Harriet Lane Home of the Johns Hopkins Hospital and the Department of Pediatrics, Johns Hopkins University, Baltimore, Md.)

Since "incidence" in tuberculosis may be subject to different interpretations, we may proceed on the premise that a positive tuberculin reaction is the surest proof we possess of tuberculous infection, although there may be a variable relationship between a tuberculous infection and actual clinical morbidity from tuberculosis. The pre-school child may be designated as from two to five years of age. Infancy, then, would comprise the first year.

It is true that, owing to technical factors, insufficient dosage of tuberculin or lowered allergy, an individual may harbor active infection and at the same time prove to be tuberculin negative. In general, however, such exceptions are not numerous in any properly conducted study.

To secure an accurate index of the incidence of tuberculous infection in children under school age in any given community, to say nothing of the country at large, it would be necessary to apply the tuberculin test to all infants, regardless of social status, and to repeat the test at least at yearly intervals, until school age is reached. Obviously such procedure at present is quite impracticable in communities of any size.

Sectional studies in a city, also, are unreliable. For example, Chadwick found in a city of 60,000 inhabitants that positive tuberculin reactions among school children varied, in different districts, from 11 per cent to 60 per cent. Comparable differences probably existed among infants and pre-school children.

In small communities health surveys made to include practically all the infants and children are quite feasible. For example, in the oft-quoted Framingham (Massachusetts) demonstration, in 1917,

* Read April 6, 1932.

366 children were tested during the first five years of life. The percentage of positive reactors increased from 15 in the first year to 52 in the fifth. The average for five years was 30.3 per cent.

The report of Hetherington and his collaborators on the incidence of tuberculosis among the school children of Philadelphia contains a few figures in the pre-school age. Of 114 children tested during the first five years, 37.7 per cent were tuberculin positive. The fallacy in accepting such results is that not all of the children in the pre-school age group were tested. This applies to practically all of those studies on school children, which also include the pre-school child.

A recent survey of a small industrial town in Tennessee included some 90 per cent of the children two years of age or younger. One hundred and twelve white children were tuberculin tested: 28 of the 112 had had no ascertainable contact with tuberculosis in the home. Of these 28, 10.7 per cent gave positive reactions. Sixteen children had been definitely exposed to tuberculosis in the home. Of these 16, 31.2 per cent gave positive reactions. Including the latter group, in 84 of the 112 children there had been definite evidence or strong suspicion of some exposure to tuberculosis. Of these 84, 28.5 per cent were tuberculin positive.

The literature contains many references to series of tuberculin tests performed on groups of hospital or clinic patients. One of the most extensive is that of Charles Hendee Smith in the Children's Medical Division of Bellevue Hospital, New York, from 1921 to 1928.

First year of life	2407 tested	5.27 per cent tuberculin positive
Second year of life . . .	1088 tested	12.50 per cent tuberculin positive
Third, fourth and fifth year of life	1527 tested	15.80 per cent tuberculin positive
Total	5022 tested	13.90 per cent tuberculin positive

It may be noted that among this hospital clientele, coming for the most part from the worst residential section of New York City, the total percentage of reactions in the pre-school age was less than half that found among the children of Framingham (13.9 per cent to 30.3 per cent).

Including other figures from hospital studies, apparently the percentage of tuberculin-positive reactors in most communities will vary from 5 to 15 in the first year to from 15 to 35 at the end of five years.

In statistics on mortality, death-rates are computed in age groups after the first year. It is not known what, if any, definite relationship exists between infection and mortality, but the figures for mortality in the United States registration area are extremely interesting. In the year 1900, deaths of infants from tuberculosis per 100,000 were more numerous than at any other period of life. In 1924 the infant death-rate had fallen below that of every age group except that between one and fifteen years. In other words, while the general decline in the death-rate had been 55.7 per cent, that for infants under one year had fallen 73.1 per cent.

Conditions Under Which Infection Occurs. In discussing the contagion of tuberculosis we may assume that the pulmonary form of the disease is practically the only transmissible type. The consensus of opinion favors the belief that pulmonary tuberculosis almost always is an air-borne infection.

Direct infection of the lung through the inspiratory air current presumably can occur when the infecting agent coughs into the radius of the inspiratory air current of the individual to be infected. Pflugge's droplets of sputum, emitted during talking as well as in coughing or sneezing, must play an important rôle when we consider the inevitable tendency of all admirers of the baby (especially the mother) to talk directly into the baby's face. In the first months of the baby's life this usually means talking directly over the baby as it lies helpless to avoid it. If the mother has active pulmonary tuberculosis, we would expect that the infant hardly could escape and that the percentage of infections would be greater in the first year than in any other. We do not have extensive statistical data on the age when primary infection occurs most frequently. A child, for example, found to be tuberculin positive at five years of age may have acquired the infection in any one of his five years of life. From incomplete figures, not yet ready for publication, it appears that the second year, when the infant is less dependent on the mother, may exceed the first, in the number of primary infections. Other figures from the same source show that the tuberculous father infects the baby in the first year almost as often as the mother, so that the intimate maternal care cannot be the sole source of these early infections.

Infection also probably reaches the lung indirectly, through contamination of mouth secretions resulting from the act of kissing or putting infected fingers or articles into the mouth. Obviously this must occur frequently throughout childhood, possibly more often

than does direct infection from a cough. It was one of the main contentions of those who believed in the theory of pulmonary involvement resulting from infection by way of the abdominal lymphatics that contamination of the mouth would result chiefly in infection following deglutition.

Cobbett's experiments, however, would seem to explain the mechanism of direct inhalation of mouth bacteria. Immediately after placing a pure culture of Bacillus prodigiosus on a rabbit's tongue, the animal's trachea was completely occluded by a clamp and, simultaneously, the rabbit was killed by a blow on the head. In spite of the tracheal occlusion, Bacillus prodigiosus was recovered from the lung. Obviously the few deep inhalations taken by the frightened animal before the clamp closed, were sufficient to draw bacteria deeply into the lung. On this basis it is possible to explain a large percentage of tuberculous infections in childhood because of the tendency of the infant or child to take sudden deep inspirations during crying, shouting or active exercise. That other respiratory diseases may be acquired in the same way seems probable. Since the normal lung shows a varied bacterial content at all times the inception of actual disease would seem to depend upon individual resistance. In the case of tubercle bacilli, resistance usually is inadequate. Factors of dose and virulence of course must play an important part.

That the home which harbors a case of active tuberculosis is by far the most dangerous environmental factor in infection is well established.

On the basis of a follow-up study in the School of Hygiene, Johns Hopkins, of 50 white families containing one or more open cases of pulmonary tuberculosis, it was estimated that, given the more or less constant presence in the home of the infecting agent, babies of such households would show 75 per cent of infections at one year of age, 90 per cent at five years of age and 95 per cent at twelve years. One might proceed, therefore, on the theory that if all children under school age could be brought up in a home free from tuberculous infection, a majority of childhood tuberculosis could be prevented. Many children are found to be tuberculin positive, however, in spite of the fact that the most careful scrutiny, including actual examination of the supposedly healthy householders, fails to reveal any infecting source in the home. In the case of preschool children at least, when the ordinary questioning as to other sources of infection fails to reveal any contacts, it is noteworthy

that repeated questioning sometimes will enable the mother to recall a chance visit from relatives or friends who can be proved to have been tuberculous. Only too frequently such visitors punctuate the rarity of the event by making a special fuss over the child. Pflugge's droplets and direct mouth infection under such circumstances can hardly be avoided. In a residual percentage of cases there is no evidence obtainable of any contact with tuberculous individuals. To explain these we must fall back on the biological characteristics of the tubercle bacillus. These organisms may retain their viability for many hours under favorable conditions and we are apt to forget the importance attached to house dust by the earlier writers in tuberculosis. Cornet insisted particularly on the ease with which such dust can be inhaled, while mouth infection must be of frequent occurrence. It requires no stretch of the imagination to realize that infection of house dust from city streets can occur only too easily. It is noteworthy that these "casual" infections, as Krause calls them, as a rule are less apt to lead to virulent disease than are the infections from open cases in the house.

To return again to the home infections. Important variations depend upon the degree of care taken to protect the child. For example, a tuberculous mother of fair intelligence, and with any degree of information as to the contagious nature of tuberculosis, is probably much less dangerous than a grandparent, suffering from chronic but often unsuspected tuberculosis, who insists on kissing the grandchild when the mother's back is turned. Fathers, also, are less apt to exercise care than mothers, while ignorant hired nurses may be the worst offenders of all.

In the attempt to prove the exact source of infection it is customary to rely on the history as given by the mother or caretaker. To be of real value, however, statistical studies should depend upon a careful personal survey of the home and household by a trained investigator, and by adequate proof of active tuberculosis in the suspected agent. At last analysis only the finding of viable tubercle bacilli in the sputum is a sure test. Practically, however, a diagnosis made at a reliable hospital or clinic may be accepted as trustworthy. Judged by these standards, not much evidence is available, but the figures presented by Katherine Merritt may be accepted. This study was made on the home contacts of 25 fatal cases of tuberculosis which came to autopsy after hospitalization in the Harriet Lane Home.

Source of infection.	Number of cases.
Mother	12
Father	6
Other adult relatives	6
Sibling	1

In order to evaluate the factors which influence infection and to study many other phases of childhood tuberculosis, Dr. E. A. Park, in 1929, organized a clinic at the Harriet Lane Home, Johns Hopkins Hospital, for tuberculous or potentially tuberculous infants under two years of age. So far as possible these patients were to be followed throughout childhood so that in time the evolution and development of primary tuberculosis could be adequately studied. To date some 315 patients have been enrolled, but the number is too small and the time of observation too short to furnish a basis for any conclusions.

The study clearly is corroborative of the danger to the child of living in contact with open tuberculosis. Of 315 children in 82 families, 72 per cent had become infected. In 61 per cent of 31 families containing only 2 children each, both children had become infected.

Another interesting development is the comparatively high percentage of siblings acting as infecting agents.

Most statistics inculpate chiefly tuberculous adults. We have found that as high as 10 per cent of 141 tuberculin-positive children under two years had no other source of infection in their homes than one or more infected siblings. In consideration of the age of the patients, this finding is suggestive that tuberculous children in the home are more dangerous carriers of contagion than has been suspected.

DISCUSSION

Dr. Joseph Stokes, Jr.: It is a great pleasure to me to listen again to a lecture by Dr. Gittings, especially from the Clinic at the Harriet Lane Home at Johns Hopkins. I would like to divert your attention for a few moments to the Children's Hospital and the work carried on there with the help of Dr. McPhedran and Dr. Cutler. The cases I wish to present are in the age group discussed by Dr. Gittings.

We have had so far 7 cases under two years of age who have been treated with artificial pneumothorax. Six of these could be called "cured"; the seventh was almost beyond hope of recovery upon its admission to the hospital. It is at least suggestive that the treatment with artificial pneumothorax, even at this age and with cavity, can be of considerable value.

THE INCIDENCE OF TUBERCULOSIS IN INDUSTRIAL WORKERS AS REVEALED BY FLUOROSCOPIC EXAMINATION*

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Before speaking of the incidence of tuberculosis, it may be well to mention the usual case-finding methods, since the care with which this is done influences the results. Probably the least searching is the usual history and physical examination, which reveals the rather obvious type of case; the roentgenoscopic or roentgenographic methods in all probability reveal practically all of the significant tuberculosis in a group examined carefully.

At the University of Minnesota, where for the last two years a fluoroscopic examination of the thorax has been made on large numbers of college students, the percentage of active tuberculosis disclosed has been approximately 0.8 per cent. Gustave Baer reported on a series of examinations of students at the University of Munich who were examined fluoroscopically and found 0.7 per cent open and active tuberculosis, and 0.6 per cent of borderline cases of active tuberculosis. Kattentidt examined 2300 seemingly healthy German students in the same way and reported 0.5 per cent open tuberculosis and about 0.2 per cent active dormant cases. A combined German report upon the examinations of 25,000 supposedly healthy young adults, made up largely of army recruits, police recruits and students, stated that there was between 0.7 per cent and 0.8 per cent of open active tuberculosis. In these studies of males and females between seventeen and twenty-one years of age there is reported between 0.75 per cent and 1.25 per cent of open, active tuberculosis, an average then of 1 per cent.

It is of interest to note that in the groups where physical examinations have been done the number of cases reported by physical examination alone is small, and that careful fluoroscopic examina-

* Read April 6, 1932.

tion has added many additional cases over and above those found either by physical examination or history.

In our organization, when we first began making fluoroscopic examinations of the thorax as a method supplementary to the ordinary physical examination, it was not with the idea of making a positive diagnosis of intrathoracic pathology, but to select the proper cases for *x*-ray. To determine the accuracy in our examinations, 100 individuals who had a negative physical examination and a negative fluoroscopic examination were *x*-rayed and in all of these cases the *x*-ray reported a "healthy adult chest" with no suggestion of pathology. *X*-rays were also taken of 123 "suspicious" cases in which there were reported at the time of physical examination harsh breath sounds, transient râles at either or both apices, prolonged expiration, dulness by percussion or unhealthy appearance, but were considered negative by fluoroscopy. The *x*-ray Department reported 122 cases normal and 1 case of minimal tuberculosis. In 1931, 500 individuals who had had a negative chest *x*-ray two or more years previously were *x*-rayed again at the time of their annual physical and fluoroscopic examination. Comparison was made between the fluoroscopic and *x*-ray findings, all of the work done independently. There were 476 cases negative both fluoroscopically and by *x*-ray examination; 15 cases showed pathology described identically by fluoroscope and by *x*-ray; 9 cases which had a negative fluoroscopic report showed some abnormality upon *x*-ray study, 7 of the 9 insignificant and 2 were cases of minimal tuberculosis without physical signs or symptoms.

TABLE I.—CASES OF TUBERCULOSIS* DISCOVERED AMONG APPLICANTS FOR EMPLOYMENT IN HOME OFFICE, METROPOLITAN LIFE INSURANCE COMPANY. (DIAGNOSIS BY X-RAY)

Year.	Applicants.	Cases of tuberculosis. Number.	Percentage of applicants examined.
Years, 1928 to 1931	14,465	141	0.98
1928	4,405	53	1.20
1929	4,780	35	0.73
1930	3,105	28	0.90
1931	2,175	25	1.15

* Tuberculosis of respiratory system.

In Diehl's study at the University of Minnesota, after a health examination of 2500 students, there were reported 15 cases of clinically significant tuberculosis. On the basis of physical findings

alone, 3 of the 15 were discovered and subsequently *x*-rayed. On a basis of physical findings plus history, 4 would have been *x*-rayed routinely. On the basis of fluoroscopic findings alone, 13 of this group would have been *x*-rayed, and on a basis of fluoroscopic findings plus history, 14 would have been *x*-rayed. "In other words, if we had depended, as most physicians do, upon the physical examination and history as a basis for ordering *x*-ray plates of the chest, we would have missed 11 of these 15 cases of far-advanced, moderately advanced or incipient tuberculosis; while if we had relied upon the fluoroscopic examination plus the history we would have missed only 1 out of the 15."

TABLE II.—CASES OF TUBERCULOSIS OF RESPIRATORY SYSTEM AMONG EMPLOYEES OF METROPOLITAN LIFE INSURANCE COMPANY HOME OFFICE ON DECEMBER 31, 1931. BY SEX AND AGE. (DIAGNOSIS BY X-RAY)

	Males.				Females.			
		Tuberculosis cases.				Tuberculosis cases.		
			Per cent tuberculosis of employees.				Per cent tuberculosis of employees.	
Age in 1931.	Employees.	Number.	Crude.	Smoothed by age.†	Employees.	Number.	Crude.	Smoothed by age.†
All Ages:								
Crude rate	3737	233	6.25	6.25	9366	335	3.58	3.58
Standardized rate*	....	...	4.22*	4.22*	....	...	4.17*	4.17*
15 to 19	250	3	1.20	1.02	2257	9	0.40	1.26
20 to 24	839	15	1.79	2.39	3789	103	2.72	2.91
25 to 29	539	27	5.01	4.19	1162	67	5.77	4.95
30 to 34	398	19	4.78	5.44	673	48	7.13	6.26
35 to 39	414	31	7.49	6.50	465	23	4.95	6.39
40 to 44	386	25	6.48	7.56	385	28	7.28	6.70
45 to 49	350	34	9.71	8.96	263	21	7.99	7.46
50 to 54	224	22	9.82	11.22	199	15	7.54	8.69
55 to 59	162	28	17.28	14.84	130	16	12.31	10.35
60 to 64	124	20	16.13	18.07	43	5	11.63	12.76
65 to 70	41	9	21.95	21.45				

* Percentage of *x*-ray diagnosed tuberculosis at all ages if age distribution of employees in each sex class were the same as for employees as a whole.

† Smoothed graphically by age.

In a personal communication just received from Dr. Willard B. Soper, medical director of the William Wirt Winchester Hospital of West Haven, Conn., and from which I am generously allowed to quote, Dr. Soper tells me that in examining a group of university students in the college year 1930–1931, routine chest films were

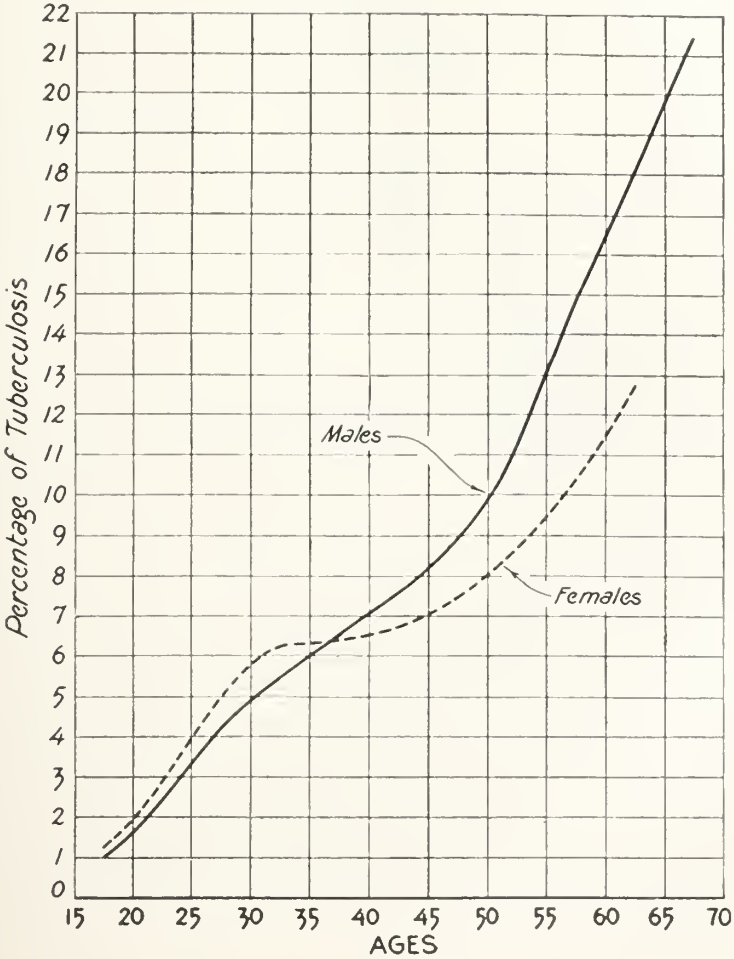
taken of 1600 students, 6 of whom showed manifest tuberculosis. In 1931–1932 in essentially the same number of students there were reported 11 manifest cases. In this series of routine *x*-rays there is an incidence of manifest and significant clinical pulmonary tuberculosis of 0.53 per cent, which compares quite exactly with the survey made by Diehl at the University of Minnesota.

TABLE III.—PERCENTAGE OF CASES OF TUBERCULOSIS OF RESPIRATORY SYSTEM AMONG EMPLOYEES IN HOME OFFICE, METROPOLITAN LIFE INSURANCE COMPANY, ON DECEMBER 31, 1931

(*Diagnosis by X-ray*)

MALES AND FEMALES—*By Age*

SMOOTHED DATA



I am not for a moment supporting the position that a fluoroscopic examination enables a person to make a diagnosis of tuberculosis, but am, rather, taking the stand that in a case-finding survey a careful fluoroscopic examination done as an adjunct to the usual physical examination does enable one to detect pathology which may be missed easily by a physical examination. It is our experience that we *x*-ray about 10 per cent of all of the cases which are examined physically and fluoroscopically, and it is our belief that we *x*-ray the proper 10 per cent now, while previously we used to *x*-ray a larger percentage of cases and were disappointed by the disproportionately large number of negative *x*-ray reports.

TABLE IV.—NUMBER OF NEW CASES OF TUBERCULOSIS* DISCOVERED AMONG HOME OFFICE EMPLOYEES OF METROPOLITAN LIFE INSURANCE COMPANY. (DIAGNOSIS BY X-RAY)

		New tuberculosis by *x*-ray in specified year.		New tuberculosis by *x*-ray in year on sick leave.	
Year.	Employees.	Number.	Percentage of employees.	Number.	Per cent of employees.
1927	11,192	138	1.23	86	0.77
1928	11,530	152	1.32	53	0.46
1929	11,966	96	0.80	29	0.24
1930	12,468	73	0.59	36	0.29
1931	13,081	67	0.51	38	0.29

* Tuberculosis of respiratory system.

To sum up this phase of the discussion then, I believe that an estimation of the incidence of pulmonary tuberculosis made on large groups of individuals examined fluoroscopically by careful workers will be much more nearly accurate than an estimation of the amount of tuberculosis as evidenced by history and physical examination, and is nearly as accurate as any other estimation which we have at the moment.

With this much foreword, our own experience in examination of applicants for employment is next reported. We began our work in the fall of 1927, including as part of the physical examination of applicants for employment at the home office a fluoroscopic examination of the thorax. The results of this work are given in table form, and as you can see readily the total number of applicants examined from 1928 through 1931 has showed an incidence of 0.98 per cent of definite pulmonary tuberculosis, the majority of the cases being clinically significant. This group is composed of about 95 per cent girls and 5 per cent boys, the majority about eighteen years of age.

Beginning in the spring of 1928, as a part of the usual annual examination of all of our home office employees, a fluoroscopic examination of the thorax was made routinely. There are at the home office 13,000 employees who have this examination each year. About 55 per cent of the entire group is made up of young women and young men of not more than twenty-five years of age, and the remaining 45 per cent is made up of both men and women up to and including age sixty-five female and age seventy male. Since the fluoroscopic examination is made upon all of our home office employees annually, repeated screenings certainly bring to light most of our cases of tuberculosis. And when we estimate the incidence of pulmonary tuberculosis among our home office employees we find the startling figure of 4.3 per cent.

On January 1, 1932, we had in the home office group 568 cases of known pulmonary tuberculosis. All of these cases are under the careful observation of our medical division, and there is assigned to this group alone one physician who is experienced in the care of tuberculous individuals. Some of the cases (15 per cent) are active and are curing at the company sanatorium or under the supervision of their own doctor. Some (45 per cent) have returned to work under observation after curing, and it is interesting to note that about 40 per cent of the employees under observation have never been to a sanatorium and have never realized that they must have had at some time an active lesion. These cases were discovered in the course of our routine annual examination and the *x*-ray showed unmistakable lesions, most of them showing definite calcification and fibrosis.

We have given serious consideration to this phase of our problem and have asked the question, why in an older group of the population is there such a large percentage of lesions as demonstrated by *x*-ray examination? We cannot evaluate accurately the rôle of labor turnover, but we believe that it is similar among our tuberculosis cases to that of the whole company, so there is not in all probability a tendency toward sedimentation of this type of employee. A great deal of emphasis has been placed on the statement that pulmonary tuberculosis is very largely a disease of adolescents and young adults, and, as previously described, about 1 per cent of the population is supposed to be diseased. Here, however, in a large group of older individuals we find nearly four and one-third times as much tuberculosis. Why? Does a large percentage of active tuber-

culosis develop and run an unrecognized chronic course in adults? Is there a reactivation in adult life of healed infantile and childhood lesions, which some persons believe actually render some degree of immunity? Do we have a residue from a heavily infected general population which had a very high death-rate twenty-five years ago? The truth probably lies largely in an affirmative answer to all of these questions.

DISCUSSION

Dr. E. P. Pendergrass: I have listened to Dr. Fellows' presentation with a great deal of interest. His observations, as presented tonight, supplement those reported by him in 1928. Briefly summarized, the fluoroscopic examination in his hands was more accurate than the most careful repeated physical examination. I am prepared to accept his conclusions and in general such observations may be true provided the fluoroscopist adheres to the following methods of examination.

1. The eyes should be well accommodated. Thirty minutes usually suffice, although some individuals require as much as one hour's sojourn in the dark. It should be remembered that individuals coming from the bright sunlight require longer periods of time to accommodate. Certain types of fluoroscopic room illumination tend to prevent entire accommodation.

2. The patient should be examined in the erect and recumbent postures, in the antero-posterior and postero-anterior lateral oblique positions, and finally rocked to one side and then the other. These observations should be made during quiet, forced and grunting respirations.

3. A fine focus tube such as the 10 milliampere Coolidge tube gives the best detail.

4. The fluoroscopic screen should be of the best and the type, whether blue or green illumination, adjusted to the individual fluoroscopist.

5. It should be possible to vary the milliamperage and the voltage according to the thickness of the patient.

With such a routine, fewer lesions will be missed, but even with the most careful technic it is surprising what large lesions it is possible to overlook by fluoroscopy. I have failed to detect a clinically diagnosed diaphragmatic pleural collection that subsequently had 500 cc. of fluid drained from the pleural cavity. Not infrequently, small soft tuberculous lesions and pneumothoraces are missed by fluoroscopy. I am certain that this has been an observation of all radiologists who are making routine films of the chest following fluoroscopy.

The ideal chest examination should include a careful physical, fluoroscopic and radiographic examination. This type of examination will become imperative in those industries in which dust is a hazard. It has been shown by Gardner in this country and a number of workers in England,

Australia, Canada and South Africa, that certain dusts which contain silica and silicates may reactivate quiescent tuberculous foci or by pathological changes in the lung create media in which tubercle bacilli grow more readily. The roentgenogram offers a permanent record by which small changes in the progress or retrogression of the lesion can be detected. I feel convinced that if routine roentgenograms were made in addition to the examination employed by Dr. Fellows, that a similar difference would be found between fluoroscopy and roentgenography as has been shown between physical examinations and fluoroscopy.

THE OCCURRENCE AND PROGRESS OF TUBERCULOUS LESIONS IN SCHOOL CHILDREN AND ADULTS*

By F. MAURICE McPHEDRAN, M.D.

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It has been shown that the tuberculin reaction increases steadily from infancy to early adult life. Running somewhat parallel with this there appears an increasing number of lesions in the lung due to tuberculous infection. The most common is a small subpleural nodule, which may appear anywhere from the apex to the base. These lesions are rarely visible in *x*-ray films except when calcified. They occasionally can be shown in the caseous state as a small, ill-delimited density; then as a circumscribed, rounded mass without marked density, which later goes on to calcification and is readily perceived provided it is not obscured by dense structures such as rib, heart or the diaphragm. Of course, many of these are lost in the roentgenogram, particularly when they are situated in the axilla, behind overlapping ribs, or on the diaphragm or toward the mediastinum. There are no symptoms or physical signs due to the lesion. This is by far the most common lesion of tuberculous infection and, except for a caseous unencapsulated focus in an infant, is of no clinical significance. In association with the pulmonary nodule, and usually more conspicuous in the excised lung, there are tuberculous changes in the lymph nodes of the hilum, which will be discussed later.

Unstable or progressive consolidation of a lobe or wedge, the common result of heavy infection in an infant, occurs infrequently in older children, except those of the Negro race. These consolidations are irregularly wedge-shaped with a flocculent periphery. The consolidation may be homogeneous, but is more likely to appear unequal, due to the superimposition of scattered bronchopneumonic patches as the lesion decreases from consolidation into normal lung. The site of these lesions, like that of the pulmonary

* Read April 6, 1932.

nodule, is anywhere in the lung, although they are more common in the upper half. Consolidation rarely extends into the extreme apex, but is more likely to be intense anteriorly at the levels of the second, third and fourth interspaces, or posteriorly at the levels of the fifth, sixth, seventh and eighth interspaces. These lesions are subject to excavation, the cavity usually being central within the mass of consolidation. The excavation is round, sharply defined, and is rarely multilocular with this type of lesion. In rare cases when the consolidation is in the lower half of the lung, its maximum density is in rare cases on the diaphragm, but more often it decreases toward the diaphragm, with cavity several centimeters from the diaphragm if excavation occurs. These consolidations may progress steadily until the lobe, or more than one lobe, is involved. On the other hand, they may spread into the opposite lung quickly by way of the air passages. There is usually marked associated enlargement of the lymph nodes, but this may be obscured when the pulmonary involvement reaches fully up to the mediastinum and extends both anteriorly and posteriorly to the midfrontal plane.

The associated lymph node lesions in their uncalcified stages are best seen in oblique and rotated exposures. The oblique film is taken with the patient turned so that the midfrontal plane forms an angle of approximately 45 degrees with the plane of the film. Rotated exposures are taken at various angles of the frontal plane with the plane of the film. When the whole lesion is shown in profile the hilum and any demonstrable enlargement of the nodes will appear at the apex of the pyramid of infiltration. Of course, when a whole lobe is consolidated the hilum is concealed.

The demonstrable lesion in the hilum in the living may be single or multiple enlarged nodes. Very often there is a marked widening of the mediastinum to the right and to the left of the spine, somewhat rounded or scolloped, with large protuberances against the pulmonary parenchyma. In infancy there are almost always associated caseous lymph nodes whether demonstrable or not; in older children calcified or uncalcified lesions of the tracheobronchial lymph nodes may or may not be recordable. These consolidations are often preceded or accompanied by a diffuse infiltration of childhood type, to be discussed later.

Mention has been made of the characteristic spread of progressive consolidations. Not infrequently there will be increase for a period of months, only to be followed by continuous retrogression with

clearing from the periphery. The lesion becomes smaller and retrogresses both toward its centers of necrosis and from the axilla toward the midline. If the lesion is bounded by an interlobar fissure, it clears toward the fissure as well as from the axilla toward the midline. The last density is commonly anterior in the second and third interspaces, or, if posterior, occurs at the level of approximately the sixth, seventh and eighth interspaces. In this site the lesion overlies the hilum shadow in films made in the usual posteroanterior position. Superimposed upon the hilum shadow, the lesion has often been described as situated in the hilum, but actually it is based on the peripheral pleura whether anterior or posterior. In the last stages of clearing there may remain only a few heavy, dense strands with a few flecks of calcium, or there may be several scattered spots of calcification. As time goes on, the spots of calcification draw together and from an area of disseminated calcium as much as 2 or 3 cm. in width, there may remain only a small spot, about 0.5 cm. in diameter.

Both the evolution and retrogression of the lesion may be unaccompanied by symptoms or signs. When present, these may be trivial. Cough and expectoration, slight elevations of temperature, are common when the consolidation is increasing. The symptoms may cease shortly after the lesion has reached its maximum, or there may even be no symptoms at any time during its course. The child remaining in bed may show no elevation of temperature during the development of an extensive infiltration. There may be neither symptoms nor definite physical signs with lesions occupying as much as a third of the upper lobe. If sputum can be obtained it is frequently positive for bacilli only on animal inoculation; when there is a definite cavity increasing in size the sputum is usually positive in smears.

Other evidences of the instability, that is to say, of the anatomical change of these and other lesions, may be found in the white count. In the progressive stage the polymorphonuclears are elevated relatively and usually absolutely. There may be an increase in the number of monocytes. In the retrogressive stage the polymorphonuclears decrease in number both absolutely and relatively, with a rise in the lymphocytes. The sedimentation-rate furnishes another test of value. It is often markedly increased and may remain so during the retrogression of the lesion when the child is doing well.

Lesions such as these usually can be shown to have developed

from close association with a case of sputum-positive tuberculosis within the household. Many of them are discovered by routine examination of contact children. As has been pointed out, they may be continuously symptom-free throughout their course. The prognosis is uncertain until this type, which is always of potentially serious import, is differentiated from the benign consolidations next described.

There is another, less common type of consolidation, which has a different appearance and a different course from the unstable lesions just discussed. They are among those described in the literature as epituberculous in character. The term is not, we think, a particularly happy one. Like the progressive consolidations, they are wedge-shaped, with the base on the pleura and the apex toward the hilum; but they are more symmetrical and more sharply delimited and they are homogeneous in character. The consolidation is densest in the center or, if it abuts on the interlobar fissure, it is densest on the fissure and on the peripheral pleura. Its density decreases by imperceptible gradations into normal lung tissue, with no flocculence at the periphery.

This type of consolidation rarely causes any disturbance of health, and elevation of temperature has been trivial in our cases in school-age children. In 1 case, a child who had formerly had enlarged uncalcified lymph nodes, later showed lymph-node calcification, which was followed by the development of one of these homogeneous consolidations. The lesion appeared without symptoms and with trivial signs, including diminution of the breath sounds and slight impairment of resonance. In other cases there has been slight accentuation of breath sounds.

These lesions are benign. Unlike the progressive consolidations they do not spread from their periphery, but they are characterized by continuous decrease. Anatomically their course resembles the former in that clearing is from the periphery toward the center of the lesion, from the axilla toward the midline; but when they clear they leave behind only a few light strands instead of spots of calcification.

Although the lesion has well-defined clinical characteristics—slight symptoms and signs—and definite *x*-ray appearances—homogeneity and sharply defined pyramidal forms—it is rarely safe to make a diagnosis of a benign consolidation without serial films. In these, as in views taken at different angles, it will be found that

there is no uncalcified enlargement of the associated lymph nodes. When there are uncalcified lymph nodes large enough to be shown in the roentgenogram, the consolidation is not of this benign type and, no matter what its temporary course, should be regarded as a definitely dangerous lesion, capable of swift and disastrous relapse. With the benign consolidations there is characteristically no demonstrable associated lesion, although sometimes calcified nodes are observed.

The group of children in whom these consolidations were first described by Eliasberg and Neuland were infants with extrapulmonary lesions. In our series they have appeared only in children of school age.

The next lesion to be considered is a diffuse flocculent or mottled infiltration, a scattered tuberculous bronchopneumonia. In the *x*-ray film the spots comprising the lesion may be light and almost imperceptible, or they may be dense, ill-delimited and almost confluent. If the lesion progresses, it increases by confluence of the spots and may excavate. In such cases excavation, as with the unstable or progressive type of consolidation, is in the upper or middle third of the lung, rarely in the lower third, and hardly ever close to the diaphragm. This distinguishes these lesions from the nontuberculous lesions, bronchopneumonia, etc., of childhood. Like the other tuberculous lesions that have been described, these have their maximum density on the pleura, usually anteriorly or posteriorly but not infrequently in the axilla. They conform roughly to the wedge shape. In clearing there may remain behind scattered spots of calcium or only a few dense spots. Gradually the strands may disappear and the spots may be drawn together, so that calcium scattered over an area of 3 or 4 cm. after some years may appear in an area of 0.5 to 1 cm. Enlargement of the draining lymph nodes is fairly common, but very often much less conspicuous than with the unstable or progressive consolidations described above. Such nodes are usually calcified in older children; uncalcified lymph nodes are rarely demonstrable, occurring chiefly in infants and colored children, and when multiple are of grave significance.

This diffuse infiltration is much the most common serious tuberculous lesion of childhood. The prognosis is, however, favorable unless an increase in density develops with confluence of the spots into consolidation. In most cases the lesion is discovered incident-

ally in serial examinations of contact children. It rarely causes symptoms or signs except with marked enlargement or confluence of the spots. Positive sputum is less common than with the unstable type of consolidation. Physical signs are commonly only slight impairment of resonance or changes in breath sounds, whether slight increase or slight decrease. As the lesion clears sometimes râles are heard, but often are not present even at the height of the lesion. Many of these infiltrations are properly classified as latent throughout their course.

Miliary tuberculosis develops from a preëxisting lesion of the lung or associated lymph nodes, or some other organ. Not uncommonly the original lesion in the lung is an area of consolidation with a central excavation; in other cases the lymph nodes are enormously enlarged in association with a small nodule in the lung. When the miliary infiltration is first discovered scattered uniformly throughout the lung from the apex to the base there may be trivial symptoms or none. The spots may be very small, almost imperceptible, when first observed in an *x*-ray film. The evolution of the lesion may be very rapid and the spots may enlarge conspicuously within a shorter time than two weeks. In children past the age of infancy this lesion is more common in the colored than in the white race, corresponding to the greater liability of the Negro to pulmonary consolidation and severe lesions of the lymph nodes of the hilum and mediastinum. Perhaps the most characteristic evidence of miliary tuberculosis is dyspnea and slight blueness of the face. Physical signs are not characteristic; there may be scattered râles; some have described a generalized hyperresonance note. In general the symptoms are those of the associated pulmonary lesion or of meningeal deposition of tubercles.

Lesions of the lymph nodes of the hilum and mediastinum are secondary to lesions of the lung parenchyma. Examination of postmortem material has given conclusive evidence that the pulmonary lesion is primary and can be demonstrated in the great majority of cases. The pulmonary lesion is often easily overlooked without *x*-ray examination of the inflated excised lung because a very small nodule, situated anywhere beneath the pleura of either lung and sometimes obscured by dense structures, may cause large lymph nodes in the hilum, which because of the circumscribed area in which they occur are not readily overlooked. When the lesion in the pulmonary parenchyma is small there may be enormous enlargement

of the lymph nodes of the hilum or mediastinum without causing symptoms or physical signs. Enlarged lymph nodes in the hilum may be completely concealed by the network of vascular shadows in this region, but when greatly enlarged they can usually be detected in x-ray films, appearing as rounded masses projecting into and contrasting with the pulmonary parenchyma. The diagnostic criteria for their recognition have been discussed elsewhere. They are chiefly significant in the uncalcified state as an evidence of recent and severe infection. Their presence is warrant to keep the child under close observation, and in many cases when the enlargement is widespread and severe, for rest in bed, because of the danger of extrapulmonary tuberculosis by metastasis from the lymph-node lesion. It is not uncommon to have the lymph-node lesion shrink so that it is no longer perceptible until calcified, and about this time there may be evident a development of an extrapulmonary lesion, such as one of the spine or of a joint.

Opie has shown by postmortem examination that in urban populations in America tuberculous lesions are common in the lung and the associated lymph nodes but are rarely found in the mesentery. It may, therefore, be presumed that a positive tuberculin reaction in most cases is evidence for a pulmonary lesion, usually the subpleural nodule, with some enlargement of the involved hilum nodes. The vast majority of these lesions are not perceptible by radiographic or other examination. It is only when an uncalcified lymph node is sufficiently enlarged to project into and contrast with the parenchyma that its presence can be detected in x-ray films. Both those nodes that are large enough to be recorded radiographically in their uncalcified stage and those that do not project beyond the hilum shadow may be perceived when calcium salts have been deposited within them. Such lesions are more common in children of contact families than in those who have no contact in the household with pulmonary tuberculosis. Calcified lesions of the tracheobronchial lymph nodes are probably, therefore, chiefly significant as evidence of a heavy first infection, suggesting that one should determine whether exposure to contagion has been or is being carried over into adolescence.

Apart from infancy, it is during adolescence and early adult life that prolonged, intimate exposure to tubercle bacilli causes its most severe damage. About the age of puberty there begin to appear the pulmonary infiltrations typical of tuberculosis in adults of the white

race. In our experience in the serial examination of children living in household contact with a case of sputum-positive tuberculosis, these lesions, developing in the upper part of the lung above the level of the posterior fifth rib, are usually first seen in the extreme apex, somewhat laterally to the midline. They are roughly wedge-shaped, based on the axillary pleura and extending downward along the axilla. From their base on the pleura they spread into the lung and, usually after a period of months or even years, a central excavation may develop. These lesions remain latent throughout a large part of their course. Sometimes they may be symptom-free and produce no physical signs for as much as four and a half years or longer. During this period a latent lesion may increase continuously or it may fluctuate in intensity and extent. Again, the lesion may develop from its first appearance to clinical tuberculosis within a period of eight months or less. In such cases it may, and usually does, become moderately advanced in extent before symptoms appear.

In general the occurrence of symptoms and physical signs is often delayed until after the appearance of excavation. Many apical lesions, including those just described, increase in extent to moderately advanced lesions, as defined by The National Tuberculosis Association, without causing either symptoms or demonstrable physical signs, whether changes in voice sounds, impairment or râles. Sometimes such a lesion will then retrogress until nothing remains but a few strands and perhaps a few spots of calcification, at which time râles may develop. In the majority of cases that go on to clinical tuberculosis I have found that râles can be heard before symptoms appear. Changes in the percussion note and in voice sounds are less easily detected in the early stages of apical pulmonary infiltration.

In the roentgenogram, the first densities are usually a few spots or a few light salients, or wedges, based on the pleura, with the apex directed toward the center of the lung or hilum. Increase in the lesion takes place by increase in the size of the spots and in the size of the salients; finally there appears the confluent shadow resulting from a merging of the various densities. After the appearance of symptoms or signs, spread may be gradual or rapid. The further evolution of apical lesions is too well known to need comment.

It may be pointed out here that during early life, that is, during adolescence and before, say, the twenty-fifth year, the anatomical appearance of the lesion is no warrant that it will not progress later

and cause a fresh increase in the lesion and fresh symptoms. Flocculent or soft spots within the apex are, of course, always evidence of a dangerous lesion, one that, on the one hand, is capable of rapid progression, and, on the other hand, will usually retrogress slowly. Again, sharp strands and salients in the apex in children or young adults are also capable of rapid spread, sometimes developing in from four to six months under conditions of severe strain, such as overwork or insufficient food.

For the supervision of soft, flocculent, apical infiltrations closely spaced serial roentgenograms are necessary. Such a lesion may heal very slowly even with rest in bed. The fact that the patient is gaining weight and has neither symptoms nor elevation of temperature or pulse-rate gives no assurance that the lesion may be safely neglected. The fundamental consideration is to watch the retrogression of the lesion until it is composed solely of sharp strands and a few spots of calcium, and to observe that it remains in this condition, sharply defined, for a period of not less than nine months before it may be considered stable enough to bear the stresses of normal living.

With the exception of the nonapical pulmonary lesions of childhood, tuberculous infiltrations rarely offer difficulties in differential diagnosis from nontuberculous pulmonary lesions. Lesions in the upper third or upper half of the chest due to tuberculosis are usually far more extensive than the symptoms would suggest. They are indolent, develop slowly and retrogress more slowly. The nontuberculous lesions are usually in the lower third of the lung, close to the heart and upon the diaphragm. Tuberculous lesions are practically always based on the peripheral pleura, suitable roentgenograms showing the profile of the lesion with its base on the pleura and its apex directed toward the hilum. The nontuberculous lesions, on the other hand, cling to the cardiac contour in the film or spread out along the diaphragm. They are much more volatile, appear much more rapidly and disappear more rapidly, with the exception of the chronic relapsing pneumonias, which have been discussed elsewhere. Sputum is negative, with persistent recurrent cough. The tuberculin reaction is negative in young children, and if negative or weak at any age is significant in differential diagnosis.

DISCUSSION

Dr. H. W. Hetherington: I am sure that we are all very much interested in Dr. McPhedran's paper on various types of tuberculous lesions in children and adults. There still remain two theories regarding the development of adult tuberculosis. Of these, the belief that adult tuberculosis commonly results from exogenous reinfection has been gaining ground. There are, however, many clinicians who believe that apical tuberculosis develops much more frequently in individuals who show childhood type of tuberculosis, namely calcified pulmonary nodules and calcified lesions of the tracheobronchial lymph nodes. During the past year, I have had the opportunity of reëxamining 112 adolescent children who showed calcified lesions of the tracheobronchial lymph nodes in *x*-ray films taken in 1928. After an interval of more than three years, there was no instance in which a new tuberculous lesion had developed. On the other hand, among 75 children whose lungs had been apparently normal on *x*-ray examination in 1928, two instances of apical tuberculosis had developed both of which produced physical signs. These results suggest that apical tuberculosis is not caused directly by the dissemination of tubercle bacilli from calcified lymph node lesions and also that children with calcified lesions are not particularly susceptible to apical tuberculosis. Since the inhalation of tubercle bacilli produces tuberculosis and since tuberculosis is commonly a household disease, it is probable that a child already heavily exposed to infection with tubercle bacilli may be re-exposed, for instance, by the return of a patient from a sanatorium or the appearance of a new case in the family and develop apical tuberculosis. This probably accounts for the high percentage of cases of adult tuberculosis that exhibit also calcified lesions of the tracheobronchial lymph nodes.

EXOGENOUS TUBERCULOUS INFECTION OF ADULTS*

By EUGENE L. OPIE, M.D.

Young children exposed to contact with tuberculosis acquire the disease so readily that its relation to contagion has been fully recognized. The exogenous infection of adults, on the contrary, has long been the subject of controversy. Tuberculosis, many have maintained, does not occur more frequently in adults conspicuously exposed to the disease than in the general population. Statistics from sanatoriums and hospitals for tuberculosis have been cited to show that the disease is not unusually frequent in doctors and nurses working in contact with it, but some have assumed that suitable precautions in such institutions may be effective in preventing spread of infection.

The strongest argument urged by those who deny exogenous infection of adults is the supposed infrequency of obvious marital contagion. If husbands or wives exposed to open tuberculosis in their consorts fail to acquire infection, adult contagion is improbable. The literature of tuberculosis contains an immense collection of statistics concerning marital tuberculosis, in great part based upon the histories of patients. Some of those who have analyzed these statistics, including the well-known statistician Pearson, have reached the conclusion that there is little if any evidence that the disease is transmitted from one partner to another, and many have been so fully convinced that no tuberculosis has its origin in adult life that they maintain the belief that all tuberculosis is acquired in early childhood. Upon this assumption the disease of adult life is a continuation of an infection which has remained latent during a long period of years.

Anatomical evidence does not support this opinion. Apical tuberculosis of adults discovered in persons who die from causes other than tuberculosis, like mortality from the disease, has its highest incidence in later life when the focal tuberculous lesions of childhood are in most instances completely healed. These

* Read April 6, 1932.

lesions of childhood are usually calcified and obsolete at a time when the apical lesions of adults are relatively fresh and caseous. There is no evidence that the lesion of adult life arises by extension from the focal lesion of childhood, and in many instances the two are in opposite lungs.

Trustworthy information concerning the occurrence of marital tuberculosis is obtainable only by direct observation of the partners of those who suffer with tuberculosis. Roentgenographic examination of the chest extends very widely the field of observation, since with it pulmonary lesions accompanied by manifest symptoms and physical signs can be defined with an accuracy previously unobtainable, and the scope of such observation is extended by the recognition of lesions unaccompanied by significant symptoms or physical signs, and hence designated latent.

At the Henry Phipps Institute Dr. McPhedran and I have studied 533 married couples of whom one or both partners suffered with tuberculosis. Of these, 376 were white and 157 negro. Carefully prepared clinical histories showed which partner first developed the disease and if the sputum of these persons contained tubercle bacilli. The following table shows the occurrence of clinically manifest tuberculosis in white wife or husband after its appearance in the other partner:

TABLE 1.—THE OCCURRENCE OF CLINICALLY MANIFEST TUBERCULOSIS IN WHITE WIFE OR HUSBAND AFTER ITS APPEARANCE IN THE OTHER MARITAL PARTNER

	Number exposed.	Number not examined.	Number with tuberculosis.	Per cent with tuberculosis; with probable error.
Wives exposed to husbands having tuberculosis with:				
Tubercle bacilli in sputum . . .	145	45	18	12.4 ± 1.85
No tubercle bacilli in sputum . .	89	33	4	4.5 ± 1.48
Husbands exposed to wives having tuberculosis with:				
Tubercle bacilli in sputum . . .	70	37	9	12.9 ± 2.70
No tubercle bacilli in sputum . .	72	47	6	8.3 ± 2.19

When husband or wife first suffered with pulmonary tuberculosis and had tubercle bacilli in the sputum, tuberculosis appeared in the other partner in slightly more than 12 per cent of instances. When no tubercle bacilli were found in the sputum of the husband or wife who first suffered with the disease, its occurrence in the other partner was considerably less frequent. The absence of tubercle bacilli at the time of examination obviously does not exclude its presence at some previous time.

Roentgenological examination reveals a large number of apical lesions, often advanced, that have caused no symptoms or physical signs. Those husbands or wives who have such lesions, being unaware of their presence, are no more likely to come to the dispensary for examination than other persons who have been in contact with tuberculosis. Hence, the percentage of latent lesions obtained by examining exposed marital partners with no manifest tuberculosis is equally applicable to those who have failed to report for examination, and is in some respects a more accurate index of the incidence of infection than manifest disease. The incidence of latent apical tuberculosis in wives or husbands who have been exposed to partners with clinically manifest disease is given in Table 2:

TABLE 2.—THE OCCURRENCE OF LATENT APICAL TUBERCULOSIS IN WHITE WIFE OR HUSBAND AFTER THE APPEARANCE OF TUBERCULOSIS IN THE OTHER MARITAL PARTNER

	Number who were examined and found to have no clinically manifest tuberculosis.	Number with latent apical tuberculosis.	Per cent with latent apical tuberculosis.
Wives exposed to husbands having tuberculosis with:			
Tubercle bacilli in sputum	82	21	25.6
No tubercle bacilli in sputum	52	10	19.2
Husbands exposed to wives having tuberculosis with:			
Tubercle bacilli in sputum	24	9	37.5
No tubercle bacilli in sputum	19	6	31 6

The latent lesions that are enumerated in the table are recognized by unmistakable shadows at the apex which often extend below the level of the clavicle. They are not only more frequent but are more extensive in married persons exposed to a partner with open tuberculosis than in those exposed to pulmonary tuberculosis with which no tubercle bacilli have been found in the sputum.

In the following table the incidence of these latent lesions in husbands and wives exposed to tuberculosis in their partner is compared with that of 142 married persons with no known exposure to tuberculosis:

TABLE 3

	Number examined.	Number with latent apical tuberculosis.	Per cent with latent apical tuberculosis, and probable error.
Exposed to tuberculosis in husband or wife	177	46	25.99 ± 2.22
With no known exposure to tuberculosis	142	6	4.23 ± 1.92

The probability that this is a chance difference is 0.00023 in 100, or approximately 1 to 500,000. We are indebted to Miss Marjorie Gooch for this estimate.

Since it may be assumed that latent apical tuberculosis occurs with equal frequency in examined and unexamined persons exposed to the disease, the total estimated number of latent lesions in those with no manifest disease may be added to the number of partners with clinically recognizable tuberculosis to obtain the total number of tuberculous lesions in exposed husbands or wives. The results of this estimate are given in the following table:

TABLE 4.—THE INCIDENCE OF ADULT TYPE OF TUBERCULOUS INFECTION IN WIVES OR HUSBANDS AFTER THE APPEARANCE OF CLINICALLY MANIFEST TUBERCULOSIS IN THE OTHER MARITAL PARTNER

	Per cent with manifest tuberculosis.	Estimated per cent with latent apical tuberculosis	Estimated per cent with both forms of tuberculosis.
Wives in contact with husbands having tuberculosis with:			
Tubercle bacilli in sputum	12.4	23.1	35.5
No tubercle bacilli in sputum	4.5	18.4	22.9
Husbands in contact with wives having tuberculosis with:			
Tubercle bacilli in sputum	12.9	32.7	45.6
No tubercle bacilli in sputum	8.3	27.6	35.9

The number of married couples of the Negro race has been too small to give satisfactory information concerning the incidence of tuberculosis in husbands and wives exposed to tuberculosis in their marital partners.

CONCLUSIONS. Husbands and wives whose partners have suffered with the disease are infected five times as often as unexposed persons, when no tubercle bacilli have been discovered in the sputum of the tuberculous partner, and nine times as often as unexposed persons when tubercle bacilli have been found in the sputum of the tuberculous consort.

The frequency of infection recognizable by roentgenographic examination in wives exposed to husbands with tubercle bacilli in the sputum is 35.5 per cent; in husbands similarly exposed, 45.6 per cent.

The foregoing observations clearly demonstrate the exogenous infection of adults exposed to intimate contact with tuberculosis.

CANCER IN MAN*

By STANLEY P. REIMANN

(From the Lankenau Hospital Research Institute, Philadelphia)

What I say in the few minutes allotted to Cancer in Man is to be interpreted in the knowledge of today, and with the definite understanding that any or all of the statements may be modified at any time, in principle or in detail, by new knowledge as it is accumulated and correlated. Obviously, it can only be very sketchy and can touch only a few points of this many-angled subject. Practical and theoretical, *i. e.*, clinical and laboratory, phases will be blended, as they should be, for no one knows how soon a theoretical consideration and a laboratory experiment will be used clinically, nor how quickly a seemingly academic question can become of the utmost practical significance. And on the other hand, no successful clinical procedure can be based on false theory.

The definition of cancer for tonight is simply this: any malignant tumor. But it might include any tumor, benign or malignant.

1. It is a mistake to single out cancer in man as a separate and distinct entity. Just as man is an animal and as such is related in most of his functions to all other animals and plants as well, so are the cancers which afflict man related to the cancers which afflict all other animals and plants. Since, therefore, man, at least from the strictly physical side, is a unit in the biological system of this earth, he is a problem in biology and so are his diseases. And so information about cancer in any living thing somehow is applicable to man.

2. And, therefore, it is a mistake to single out certain peculiarities in the habits of man and make them responsible for his cancers. Thus he gets cancer because he eats canned foods or because he does not eat enough grapefruit, etc. This idea comes from loose thinking or none at all; more particularly it comes from confusing the immediate or exciting causes of cancer with the deeper, more fundamental ones. This leads to the statement that:

* Read May 4, 1932.

3. It is a mistake to say that the cause of cancer is unknown, for many causes are known. If jagged teeth and syphilis were annihilated, many cancers of the mouth would disappear; if lacerated cervices were all properly repaired, many carcinomas of the uterus would never develop. If certain occupations were properly safeguarded, another group would disappear; if all of certain benign tumors, ulcers, blemishes, etc., were removed, still another crop would never be harvested, etc.

But proper thinking groups all of these chronic irritations, using that term in its broadest sense, under exciting or immediate causes. The fundamental underlying reasons why cells should multiply as cancers after such stimuli is not known. Nor will it be known until we know much more about what makes cells multiply in an orderly manner. But then, also, in spite of the satisfaction often expressed, we know just as little about the fundamentals of other diseases. Which leads to the next statement.

4. It is a mistake to compare cancer with any other disease—except for the differences. Especially is this true of infections like tuberculosis or syphilis. Actually, although we glibly say the cause of tuberculosis is the tubercle bacillus, that of another infection is the streptococcus, etc., we know little more, if any, of the reasons why the tissues react in a certain way to the presence of these organisms than we know why the tissues react to form a cancer in the presence of an agent similarly as definite, like tar or a jagged tooth.

No one who has studied both cancers and the infections can fail to appreciate the much greater complexity of the latter.

5. It is a mistake to consider cancer as a single clinical entity. It is a disease with protean clinical manifestations. Under this name are grouped many conditions with widely different exciting causes, clinical manifestations and symptoms indicating different details of treatment. If the comparison is made at all, it should be not with one infectious disease such as streptococcus septicemia or tuberculosis, but with the whole group of inflammatory diseases. Just think of the abysmal ignorance if all inflammatory diseases were considered one and the same. From which it follows that:

6. It is a mistake to think that one specific organism causes cancer, for then its manifestations, more varied by far than any other group of diseases, would be more mysterious than ever. In spite of filtrable viruses, etc., thought to be causes of certain very special growths like chicken sarcomas, etc., if they are the causes

at all, they are the exciting ones and not the fundamental ones. From this again it follows that:

7. It is a mistake to believe that cancer can be properly controlled by the use of toxic materials such as lead, selenium, gold, etc. At most they inhibit in but few cases and do damage in many.

The ideal to be attained is to remove the exciting cause, if known, and so rearrange the internal chemistry of cells and environment that they themselves stop their own growth or guide it into useful channels. This is so complex, so intimately connected with the attributes of life itself that:

8. It is a mistake to believe that the ultimate cause and cure of cancer will be found by some amateur or dabbler, or well-meaning shoemaker, clerk, etc. "Chance favors the prepared mind," and when finally understanding is reached, it will be based on the labors of many sung and unsung workers with the patience of Job, the insight of a Bacon and the good nature of a saint working under the difficulties and discouragements of that poorly-rewarded profession, cancer research.

9. It is also a mistake to believe that a long-time practitioner can suddenly become creative in experimental cancer research. But a clinician can, should and must carry on his part of the job; not regard cancer patients as entirely hopeless, for much can be done to relieve suffering and despair, and withal, study and record every phase of the manifestations presented to him by the disease. And, of course—

10. It is a mistake to be premature in drawing conclusions, especially as to the efficacy of a remedy. Cancer is often a long-drawn-out disease and ten years' duration of untreated cancer is no rarity.

11. It is a mistake to regard morphology as the end of a study in cancer. Important as morphology is, and it is the foundation of our knowledge—would that there were more expert morphologists both of the gross and microscopic fields, competent tissue pathologists are certainly relatively few in number—morphology is, nevertheless, just the beginning of the study. Control of natural phenomena is obtained by a knowledge of the dynamics of a process, mostly of its physics and chemistry.

It is, therefore, not enough to merely look at a cancer, give it a name, describe it and then pigeon-hole it in a table of classifications. This remark is made for those who worry excessively about specific

names and is a gentle jibe at those who are cluttering up the literature and inexperienced minds with new labels for old bottles.

To pick out one last thought from the many which clamor for recognition; in a subject so long known to be vital to humankind, there has naturally been an enormous outpouring of ideas and work, so that:

12. It is a mistake to expend much energy on an idea until at least a cursory survey of cancer history has been made. To many of our forefathers, ideas came as brilliantly as to us and had they our present equipment and apparatus, perhaps, they would do better than we. It does not take a very comprehensive knowledge of music to recognize the ideas of old masters dressed up as modern jazz. Similarly in cancer, the old ones seem to be the best, judged by their popularity. Not the least harm that the old ones do when dressed up and presented as new, is to render still more unbearable the already unbearable burden of hunting through the literature for the divine spark of originality.

Finally, to remind you once more that cancer is a biological problem, and in spite of my remarks that it is not a single clinical entity, there is, nevertheless, a single phenomenon, a least common denominator in all cancers, and that is cell multiplication. If we study this we are certain of studying at least one of its fundamentals and I turn you over to a biologist, Dr. Hammett, for this part.

PROGNOSIS IN CARCINOMA OF THE STOMACH; REMOTE RESULTS OF SURGICAL TREATMENT TO JANUARY, 1930*

By ASTLEY P. C. ASHHURST, M.D.

AND

JOHN W. KLOPP, M.D.

The literature on carcinoma of the stomach is so voluminous that writers should refrain from adding to the confusion unless they follow some uniform plan of record such as that proposed by Greenough and Simmons.[1] In analyzing our cases to follow we lay no claim to special success in securing what are apparently a few cures, but present even such modest results as a challenge to a disease which, unmolested, always carries the patient to his grave with absolute certainty. If it could be shown, for instance, that a patient with cancer of the stomach may live, untreated, for five years, then our conclusions regarding the efficiency of surgical eradication would be unsound. But lacking such proof, we must rely upon the surgical method as the only successful means of treatment.†

The stomach is the most common site of human cancer. The statistics from which the diagram in Fig. 2 were drawn‡ show that there is an unmistakably significant trend toward an increase in

Figures and charts refer to cuts which will appear later in Archives of Surgery.

* Read May 4, 1932.

† We are aware that isolated instances of patients surviving over five years with untreated carcinoma are on record, but not, as far as we know, of gastric carcinoma. Dr. P. P. Klopp recalls a woman with unmistakable clinical cancer of the *left breast* in the days before the therapeutic use of radiant energy, who refused treatment, but nevertheless lived and died with her tumor after eighteen years. The longest period of survival in surgically untreated cancer of the stomach that we were able to find in the literature is three years. (Bloodgood, 1930.[2]) (Hitzenberger and Merkler, 1931.[3])

‡ The figures from which this drawing were made represent the *death-rate* per hundred thousand, and not the *incidence* of cancer.

According to the medical department of the Metropolitan Life Insurance Company, there are no reliable statistics for the incidence of cancer of the stomach, the best available figures on deaths probably being those furnished by the Annual Reports of Mortality Statistics published by the U. S. Bureau of the Census. (Personal communication.)

However, since the death-rate and incidence are so nearly alike, the figure given above portrays incidence accurately enough.

the incidence of this disease, especially among males who are apparently afflicted more frequently than females, the difference between the sexes widening with the passage of years. Caucasians suffer more often than blacks (Fig. 3), and economic station has some bearing, for the more prosperous group seems to suffer less frequently (Fig. 2). The age incidence of carcinoma of the stomach is shown in Fig. 3, for a vast series of cases. Our own figures show that of 54 cases, most, at the time of treatment, were in the sixth (18 cases) and seventh (18 cases) decades of life.

Ages of Patients at Time of Treatment to January, 1930.
(Clinic of Dr. Ashhurst)

	Cases.
Third decade of life	2
Fourth decade of life	4
Fifth decade of life	8
Sixth decade of life	18
Seventh decade of life	18
Eighth decade of life	4
Total	54

The significant particular about both sets of figures is the surprisingly high incidence in the young adult, a fact which belies the usual textbook statement. Our youngest patient was twenty-seven years of age, the next youngest being thirty and thirty-three years, respectively. While the average age of patients in our own series at the time of treatment was about fifty-six years, yet more than 25 per cent of them were aged fifty years or less. Among the reports of stomach cancer in young patients are those of Mintz's[4] eleven-year-old boy; Marble's[5] seventeen-year-old girl; Morian;[6] Jones and Scott[7] (five-year cure in twenty-four-year-old man); and Nicholson.[8] Osler and McCrae[9] collected 6 reported cases under the age of ten.

We observed more than twice as many men as women (38 male; 16 female).

Diagnosis of Cancer of the Stomach. *Symptoms.* Gastric carcinoma patients fall into three groups according to the type of clinical symptoms from which they suffer: (1) those with a short history (*i. e.*, up to six months), (2) those with a long history (over six months), and (3) those with no history of the dyspepsia syndrome.[10]

Group 1 comprises about 50 per cent to 60 per cent of all cases in our experience, a figure which agrees with those cited by Dwyer, Blackford and Turner, and by Oberniedermayr and Stahnke.[12]

About 35 per cent of all cases fall into Group 2.[10]

According to some authorities with significantly large series, Group 3 is composed of 2 per cent[10] to 18 per cent[11] of all cases. Our ward case No. 3899 is an example of one of these patients who had no complaint of digestion or stomach, showing how difficult it is, occasionally, to make a correct diagnosis.

Ward No. 3899. A woman, aged thirty-three years, came (February 17, 1925) complaining of pain in the lumbar region and the pelvis. The presence of carcinoma of the stomach was not even suspected. She was examined several times by Dr. Haines (cystoscopy), for ulcerative cystitis and died a month after admission of exhaustion and bedsores. Only at necropsy was carcinoma of the stomach (nonobstructive) found, with secondary carcinoma of the pancreas, the lungs and the ovary.

At first, most of the patients complain of indigestion and lack of appetite, especially for flesh, loss of strength and weight, rather than actual pain. Later, most of them develop pain, usually located in the epigastrium, but not necessarily so, and this occasionally radiates to the lumbar or shoulder region, or into the pelvis. The coëxistence of epigastric tenderness is common. The prominence of vomiting depends upon whether or not there is pyloric obstruction. Those cases in Group 3 (without the dyspepsia syndrome) often suffer from weakness and anemia, cardiac disturbances, fever and sweating, or from neuralgia (bone metastasis). The peculiar lemon-tint of the skin of anemic patients is said to be characteristic of gastric cancer. The difficulty of diagnosis based solely upon symptomatology is illustrated by the following 4 cases:

One woman, aged forty-eight years, who had been under the care of an able neurologist for six months, with the diagnosis of neurasthenia, was found (by exploratory laparotomy, 1925) when a surgeon's advice was finally sought, because of failing strength, anorexia and abdominal discomfort to have an operable mass of carcinoma at the pylorus. Gastrojejunostomy brought symptomatic relief, maintained until the patient's death four months later from lobar pneumonia.

A man, aged seventy-nine years, had lost 42 pounds during the preceding two years; for eighteen months his stomach had troubled him, his chief complaint being pain there three to four hours after meals, *relieved* by taking food. This is considered fairly characteristic of ulcer of the duodenum. Exploration (1919) showed a fixed, but not entirely immovable mass on the posterior wall of the stomach, much nearer the cardia than the pylorus, and causing hour-glass deformity. Gastrogastrostomy (at W. R. G. Hospital, March 14, 1919), brought immediate relief, which was maintained for eighteen months. Secondary nodules then became palpable in the liver,

and on one occasion the patient vomited some of the tumor, which was shown under the microscope to be adenocarcinoma. Death occurred nine months after operation after a terminal illness of three weeks.

Another man, aged fifty-two years (January 28, 1916), had been under treatment in the medical ward; and (just four weeks after he had been dismissed as "cured" of "ulcer of the stomach") he was readmitted to the surgical service with an inoperable carcinoma. This patient *whose gastric symptoms extended over a period of two years*, was also relieved by gastrojejunostomy (January, 1916), and returned to his work as a farmer, plowing and reaping all summer; in September he came back with carcinomatous nodules in the otherwise empty sac of an inguinal hernia. He continued to be comfortable so far as his digestion was concerned, until January, 1917, and died in February, thirteen months after operation.

Another patient, a woman lawyer, aged thirty-nine years (1916), had been complaining of stomach symptoms for two years, and during all this time had been under the care of a professor of therapeutics. When he finally asked a surgeon's opinion, exploration (November 18, 1916) showed inoperable carcinoma of the stomach with malignant ascites. Death occurred nine weeks after operation.

Precancerous Lesions. We do not wish to engage in the unseemly philippic now raging in the journals over the question of ulcer as a precancerous stomach lesion. Alvarez and MacCarty[13] are the authors of the now hackneyed quotation, "Gastric ulcers larger than a quarter (2.4 cm. in diameter) or carcinomas smaller than that are so rarely encountered that the physician with an average practice may go years without seeing one;" and MacCarty's estimate as to the high incidence of precancerous ulcer is well known, even if not acceptable to the majority of his colleagues. The English and continental authorities hold to a more conservative view, as do the majority of our own countrymen, and seem somewhat disinclined to accept all of MacCarty's histologic criteria of malignancy without question. The following two case histories suggest that though the *symptoms* of *gastric ulcer* may be relieved by gastrojejunostomy, yet carcinoma can actually develop in the ulcer or in the scar left by its healing:

Ward No. 3808. A man, aged forty-five years, came (January 15, 1925) complaining that for three months only he had had constant epigastric pain, aggravated for about two hours after eating.

Fifteen years before (1910), he had had a gastrojejunostomy performed elsewhere for ulcer of the stomach, when he was twenty-nine years of age. Exploration (1925), showed an inoperable mass on the lesser curvature near the cardia, with metastases to the right lobe of the liver, and to the abdominal wall and umbilicus. The gastrojejunal anastomosis did not admit the

finger. There was no obstruction to cardia or pylorus. Biopsy showed adenocarcinoma with great increase of fibrous tissue. The patient died four months later. If the cancer had grown at the pylorus, and not at a site where symptoms are so often delayed, the patient might have come to operation early enough for hope of cure. He certainly had had carcinoma for more than three months and for less than fifteen years.*

Ward No. 3167. A man, aged forty years, came (December 18, 1923) complaining of stomach trouble for the previous seven weeks, since when he had been having epigastric cramps.

Eleven years previously (1912), a partial gastrectomy (Polya) had been done (by Dr. Frazier, in this hospital) for a chronic ulcer on the lesser curvature which showed no evidence of malignancy under the microscope. Exploration (1923) showed a fixed, inoperable mass on the lesser curvature with metastases to the liver. Death followed in two months. No necropsy was permitted.

Ulcer is not the only stomach lesion held to be precancerous, for, as T. Grier Miller[14] points out, benign polypus, and even chronic gastritis are known to be precursors of malignancy. Sprunt[15] reports the coëxistence in the stomach of tuberculosis and cancer, but carefully avoids any interpretation of the fact.

As we have shown that to reply entirely upon symptoms for diagnosis is dangerous practice in gastric cancer; careful physicians and surgeons should take every means to investigate the possibility that their patient's symptoms might be due to carcinoma. The means of diagnosis, aside from symptoms, which have just been discussed, might be classified as follows:

I. *Purely Clinical Methods.*

(*a*) Palpable tumor.

57 per cent of our cases.

60 per cent of Oberniedermayr and Stahnke's series.[12]

A freely movable, palpable, small tumor is more of a favorable sign than otherwise (Mayo), because it indicates that the growth is accessible.

(*b*) Cachexia.

(*c*) Coffee-ground vomitus.

* Whether or not this carcinoma developed at the site of the former ulcer, perhaps in its scar, is uncertain. But as it is well known that gastrojejunostomy will not prevent the development of an ulcer in the stomach, it is not logical to expect it to *heal* such a lesion. It is our own firm belief that whenever possible an ulcer of the stomach should be treated by wedge resection or partial gastrectomy. Yet a carcinoma may develop continguous to, but separate from, a chronic ulcer in the stomach, as in the case of Ernest M., whose history is summarized on page 131. Moreover, even after a benign ulcer has been excised, carcinoma may develop after many years. (Ward case No. 3167 following.)

II. *Laboratory Methods.*

(*a*) Chemical examination of stomach contents and of stool:

1. For strength, concentration and type of acid.
2. For occult blood.

(Rutimeyer estimates that this finding is positive in 100 per cent of cases; Anschütz, 94 per cent; Boas, 95 per cent.)[10]

(*b*) Microscopic examination of stomach contents for abnormal objects. (Oppler-Boas bacilli; fragments of tumor).

(*c*) *X*-ray.

A positive *x*-ray diagnosis is nearly 100 per cent perfect, but a negative is not.

III. Unusual, new and obsolete means.

(*a*) Gastroscopy.
(*b*) Gastro-photography.
(*c*) Serum diagnosis.
(*d*) Pneumoperitoneum.
(*e*) Inflation or distention of the stomach.

IV. Exploratory laparotomy.

Exploratory Laparotomy. Exploratory laparotomy is sometimes the only means of ascertaining the truth. Nor should other means of diagnosis be persisted in too long, lest the stage of operability (even of palliative operation) pass without the diagnosis being certain. It is true that exploratory laparotomy is not infallible: among the patients formerly included in the present series of gastric carcinomas were three on whom gastrojejunostomy was done under the impression that the inoperable mass found obstructing the pylorus was carcinomatous; but as these 3 patients remained in good health more than three years after this palliative operation, they have been removed from the list of cancer patients and transferred to that of benign ulcers, thus reducing the number of carcinoma patients from 57 to 54.*

In all the other patients the correctness of the diagnosis has been proved either by the lesions found at necropsy, at operation (secondary nodules in liver, etc.), by laboratory examination of the excised specimen, or by the patient's death. In a fourth patient, partial gastrectomy was done upon a clinical diagnosis of carcinoma, but

* Schönbauer and Orator (1924)[16] report the case of one cancerous patient who survived gastroenterostomy four years, and of another sixteen and a half years; and Daneel[17] (1908) records one seven-year survival after gastroenterostomy.

under the microscope no malignancy could be found. Including these 4 patients, later transferred from the carcinoma to the ulcer list, there were 46 patients, who, when their abdomens were opened, were thought to be suffering from carcinoma. Four errors in clinical diagnosis among 46 patients is a percentage of less than 8.7 per cent. Certainly no other means of clinical diagnosis is any more accurate than exploratory operation. Furthermore, these errors are not objectionable because they represented what proved to be proper treatment in spite of the error.

X-ray. Apart from exploratory laparotomy, we believe roentgen-ray study to be the most nearly infallible means of diagnosis; and we have to thank Dr. Ralph S. Bromer and his staff for their coöperation in their conduct of these studies. Yet many patients were so ill from starvation and their clinical diagnoses so reasonably certain, that operation was done without the delay of *x*-ray. It is interesting to note in passing that the first radiogram of the stomach was made in 1896, by an American, John Conrad Hemmeter.[18]

Gastric Analysis. Though many brilliant contributions to pure science and the physiology of digestion have been made by chemists from the time of the first work on the chemistry of digestion—by John R. Young[19] in his graduating thesis from the University of Pennsylvania on "An Experimental Inquiry into the Principles of Nutrition and the Digestive Process" (1803), through Beaumont[20] to the present—nevertheless, in actual practice, we fail to lay as much stress upon chemical analysis as some authorities, and can recall no case in which such findings outweighed all other considerations. Even the demonstration of lactic acid is regarded as equivocal, as it has been proved to occur in noncancerous conditions.[21]

Treatment of Cancer. *No Operation.* In 12 of our patients no operation was attempted, 3 patients refused and in 9 it was considered useless to attempt even exploration. Only 4 of these 12 patients died in the hospital; of the 8 discharged, 3 died within less than seven weeks; 1 was still living in misery when last heard from, a year later; and 4 could not be traced.

The average duration of life from the onset of the first symptom is a little over a year (15.7 months in a vast series of cases, 1919–1927, taken in the San Francisco Cancer Survey).[22]

Operation. As was indicated earlier, there have been no known cures after treatment by electricity, light, radiant energy, drugs, chemicals or biologic products. Surgical removal of the lesion,

for the present, is our only hope. Although stomach surgery has a long history, there being a record of an operation in 1625 upon the stomach of a patient who had swallowed a knife,[23] it is only since Billroth's first successful pyloric resection, in 1881,[34] that carcinoma in this organ has been cured. This brilliant Zürich professor, later of Vienna, heads a famous list of surgeons who distinguished themselves in this field, including his pupil, Johann von Mikuliez-Radecki (the first to use gloves in surgery, cotton ones, later superseded by rubber, introduced by Halsted); Vincenz Czerny; Anton Wolfler (first gastroenterostomy); 1881 v. Haberer, Finsterer, and many others throughout the world. One of the reasons why there are no Billroths today is that there are so *many* of them. Today there are, in widely scattered regions, many surgeons capable of doing, almost routinely, what was considered most unusual by the pioneers during the evolution of stomach surgery.

Operability. If a carcinoma patient is to be cured he must belong to that class which lends itself to radical operation. In what proportion of patients with gastric cancer is there opportunity of performing a radical operation? The bar diagram in Fig. 4 shows the proportion, in per cent of the whole number of patients seen, that each type of exploratory (palliative) and radical operation bears to the other two, and to the whole number of patients observed by the surgeon. (Table 1.)

Whether or not these percentages are calculated from the whole number of patients seen, or merely from those coming to operation makes a significant difference in the result. The wide variation in statistics depends, among other things, upon this very influential factor. Careful selection of cases, with exclusion of those thought to be too risky, will reflect itself largely in a favorable influence in the radical operability column as well as in the mortality column. Unfortunately, as will be noted by a glance at Table 1, many authors reckon as 100 per cent, merely the number of patients operated upon and not the total number submitted to their judgment. Balfour,[25] for example, very modestly offers this fact, partially as an explanation of the clinic's favorable results in peptic-ulcer operations. Statistics, to be absolutely comparable, should include those patients who refuse operation or who are inoperable.

The average percentage of carcinoma patients submitted to resection in the statistics of Fig. 4 is 30, with a maximum of 46 and a minimum of 10. Up to January 1, 1930, out of a series of 54 patients

TABLE 1.—THE NUMBER OF CASES OF GASTRIC CARCINOMA OBSERVED BY VARIOUS AUTHORITIES; THE NUMBER AND PER CENT OF EACH TYPE OPERATED UPON.

Author, years.	No. of cases observed.	No. of operations.	Per cent.	Exploratory	Per cent.	Palliative.	Per cent.	Resection.	Per cent.
Alessandri (1920–1927)	83	67	81	19	28	19	28	29	43
Alexinski (1914)	....	....		....	..	....	.	297	
Altschul (1895–1911)	612	422	69	143	34	217	51	64	15
Balfour (1924–1926)	..	113	.	48	42	19	16	46	40
Bagozzi (1920–1927)	110	88	80	24	24	31	35	33	38
Bastianelli, P. (1903–1927)	374	272	73	80	29	95	35	97	36
Bastianelli, R. (1928)	....	513	..	128	23	176	32	293	44
Billroth (1878–1890)	....	....	..	....	..	28	..	28	
Bindseil (1912)	....	....	..	....	..	38			
Borelius (1898–1919)	659	501	76	125	24	229	45	147	29
Brunner (1896–1900)	47	30	64	12	40	9	30	10	33
Brunner (1896–1905)	..	62	..	12	19	32	51	18	29
Carle and Fantino (1891–1897)	136	48	35	10	20	24	50	14	29
Caspersohn (1890–1900)	54	38	70	16	42	2	5	20	50
Cheever (1913–1922)	236	100	42	24	24	53	53	23	23
Crcite (1884–1906)	241	211	88	42	19	119	56	50	23
Crossan	80	51	64	17	33	20	39	14	27
Czerny, R.	....	38	..	9	23	17	44	12	31
Danecl	395	302	76	26	8	203	67	73	24
Deaver	....	....	..	....	..	....	..	46	
De Bucle	258	247	96	....	..	51	20	197	79
Delore and Alamartine	171	158	92	19	12	96	60	43	27
Delore and Santy	113	107	95	20	18	57	53	30	28
Derjushinsky	....	146	..	72	49	16	10	58	39
Eiselsberg	....	....	..	....	..	45	6	22	3
Eljaschev	41	37	90	9	24	10	27	18	48
Eusterman and Bueerman	1408	631	45	249	38	100	16	290	46
Faroy	....	64	..	8	13	39	61	16	25
Feurer	....	....	..	....	..	....	..	58	
Finsterer	....	535	..	68	18	70	19	348	65
Frank	77	62	80	32	52	9	11	21	34
Fumagalli	228	200	88	30	15	87	44	83	41
Galpern	....	79	..	14	17	33	41	32	40
Gamerini	....	95	..	29	30	40	42	26	27
Gar	....	....	..	....	..	....	..	18	
v. Hacker	....	....	..	....	..	51	..	42	
Hanssen	280	207	74	55	26	81	39	71	34
Hitzenberger and Merkler	272	94	35						
Hoffmann	....	309	..	55	18	144	46	103	34
Kleinschmidt	106	95	90	16	16	62	65	17	17
Küttner	....	....	..	....	..	....	..	102	
Makkas	....	458	..	68	14	223	48	163	35
Mayo, C.	....	2094	..	746	35	612	29	736	35
Mikaye	....	....	..	....	..	....	..	116	
Mündler	....	20	..	5	25	11	56	4	20
Nicholson	344	185	54	82	44	59	32	44	24
Nyström	....	....	..	....	..	....	..	225	
Oberniedermayr and Stahnke	237	222	94	66	30	73	32	83	37
Persson	....	1150	..	339	29	450	39	361	31
Petersen	....	....	..	....	..	....	..	57	
Pucinelli	....	110	..	31	28	29	26	50	45
Ribeira and Sans	....	93	..	14	15	50	53	29	31
Rossi, B.	352	227	65	58	25	87	38	82	36
Rossi, F.	228	200	88	30	15	87	44	83	41
Schönbauer and Orator	432	387	90	104	26	126	32	157	40
Segre	....	34	..	3	9	21	62	10	29
Serafini	....	70	..	31	44	34	48	5	7
Sherren	....	....	..	....	..	....	..	27	
Steudel	....	60	..	8	13	45	75	7	11
Stich	159	109	68	14	12	69	63	26	23
Stumpf	49	44	90	1	2	22	50	21	47
Taniguchi	1422	1235	86	265	21	426	34	544	44
Temoin	....	336							
Vecchi	....	102	..	23	22	54	52	25	24
Volkmann	163	145	89	30	20	60	41	55	37
Walton	262	204	78	(132)	65	(132)	65	62	31
Weil	445	364	82	128	35	116	30	120	32
Ashhurst and Klopp	54	42	78	16	38	20	48	6	14

with carcinoma of the stomach under the care of the senior author (A.P.C.A.), only 6 (11 per cent) were observed early enough for radical operation. Of these 6 patients, however, 4 (66 per cent) have survived their operation for seven years or longer, and may be reckoned as "five-year cures."

Five-year Cures After Partial Gastrectomy for Carcinoma (A.P.C.A.)

1. Benjamin E., aged forty-three years, operation, 1919. Well in April, 1932 (thirteen years).
2. Ernest M., aged fifty-five years, operation, 1920. Well in April, 1932 (twelve years).
3. Harry W., aged sixty years, operation, 1925. Well in April, 1932 (seven years).
4. John D., aged sixty-eight years, operation, 1925. Well in April, 1932 (seven years).*

The percentage of five-year cures reported by surgeons may be distorted in all sorts of ways, as shown in Fig. 5 and Table 2, which are self-explanatory. The important column is *the percentage of the total number of patients under observation who obtain five-year cures:* a high figure in this column is an index of what is called "surgical judgment." (See column 2, Table 2, and the first bar in Fig. 5.) A comparison of various surgeon's results is most difficult, since some report per cent of survivors, others per cent of those appearing in the end-result clinic, etc.

The duration of life among the patients "cured" at least five years is sometimes remarkably long, considering the expectation of life at the average cancer age.† (See Fig. 6, expectation-of-life curve drawn from figures of the Metropolitan Life Insurance Company, Industrial Department, 1928, for selected ages between ten and seventy years).

* A fifth patient, John M., aged forty-five years, who was operated upon in 1921, was well in 1930 (nine years); but as there is disagreement between pathologists as to whether or not his lesion (excised from the lesser curvature) is or is not carcinoma, he is not included. Reports of cures, such as that by Quigley,[26] in which there is no reasonable proof of carcinoma, should be definitely so labeled in their titles.

† Bloodgood (1930)[2] mentions a twenty-year survival after partial gastrectomy for cancer; Pers (1928)[27] two, of twenty-two and a half and nineteen and a half years, respectively; Schwyzer (1930),[28] one of twenty-four years; and Weil,[29] two, of nineteen and twenty-one years, respectively. Of W. J. Mayo's patients (1918),[30] thirty-five lived more than six years with a gradual decline in the total number living, as shown in Fig. 6, where the data have been superimposed on an expectation-of-life curve to show the similarity in shape. Balfour (1932)[31] reports from the Mayo Clinic 128 patients who lived ten years or more after radical operation; 1 of our survivors, Benjamin E., is living April, 1932, at the age of fifty-six, thirteen years after operation.

TABLE 2.—FIVE-YEAR CURES IN CANCER OF THE STOMACH; EXPRESSED AS PER CENT OF ALL PATIENTS, ALL OPERATIONS, RADICAL OPERATIONS, AND OF SURVIVORS OF RESECTION.

	1. No. of "5-year cures."	2. Per cent of all patients	3. Per cent of all operations	4 Per cent of radical operations	5. Per cent of survivors of resections
Frank	1	1 6	1 3	4 8	
Cheever	3	1 3	3 0	13 0	15 0
Paterson	.	. .	1 4	4 7	6 5
Persson	17				
Mayo, W. J.	.	. .		26 0	
Douglas		. .	.	27 0	
Pauchet				31 3	
Mayo, C.	59			8 1	9 3
Nicholson	2	0 58	1 04	4 5	
Hitzenberger and Merkler	2	0 73	2 1		
Hoffmann	4	.	1 3	3 9	7 4
Petersen	3	. .	5 3	. .	8 3
Ribeira and Sans	5	. .	5 4	17 1	33 3
Stich	0	0	0	0	0
Stumpf	0	0	0	0	0
Taniguchi	49	3 5	4 0	9 0	11 1
Témoin	12	3 5	.	12 0	
Walton	33	7 4	9 1	27 5	33.0
Billroth	1	.	. . .	3 6	8 3
Borelius	7	1 1	1 4	4.8	6 0
Daneel	5	1 3	1 6	6.8	9 6
Derjushinsky	3	. . . .	2 0	5.2	15 8
Finsterer	50	. . . .	9 3	14.2	
Nyström	28	. . . .	. . . .	12 4	15 8
Deaver	2				
Jones and Scott	1				
Schwyzer	1				
Pers	3	. . . .	. . . .	10 0	15.0
Anschütz	20				
Küttner	27				
v. Haberer	2				
Ashhurst and Klopp	4	7.4	9.5	66.6	80.0

EXPLORATORY OPERATION. As indicated already, exploration was urged on every patient who we thought would survive. Some writers claim that the question of inoperability may be as easily decided by roentgenologic study as by exploration. This has not been our own experience. In spite of a negative *x*-ray examination, we once found a mass of carcinoma (unfortunately inoperable) high in the fundus of the stomach (greater tuberosity). Nor has a positive roentgenologic report, however unfavorable, ever deterred us from opening an abdomen in the hope of being able to do at least a palliative operation. In fact, it may be said that every one of these operations has been commenced as an exploration. Fortunately, in

62 per cent of these explorations it was possible to do either a radical operation (in 45 per cent of all operations), or a palliative operation (in 48 per cent of all operations), leaving only 16 patients (38 per cent of the total operations), in whom nothing could be done. (See Fig. 4 for a comparison with the results of other authorities.)

Mortality of Exploratory Operation. The death-rate in these 16 exploratory operations was high (43.7 per cent), and all patients who survived the exploration and were sent home, except 1, died in less than five months. The one exception is a man living, in miserable health, one year after exploration which showed a carcinoma high on the posterior wall of the stomach (fundus), not causing obstruction, but with metastases already in the liver.

The cause of death after exploration, whether the death occurred while the patient was still in the hospital or after discharge, was attributed to the progress of the disease in every case except 1; in the latter patient, pneumonia was the cause, death occurring four days after exploration under ether. If this were the only one to be recorded as a postoperative death, our operative mortality in exploration would fall from 43.7 per cent to 6 per cent, which is nearer the figures given by many surgeons under these circumstances. (Fig. 7; Table 3.) The anesthetic used in our exploratory operations was:

Ether. Three times with 2 deaths; 1 from pneumonia on the fourth day; the other from the patient's disease, after one week.

Nitrous oxide gas, twelve times, with 4 deaths, all from the patient's disease, at intervals from twelve days to six weeks after operation.

Local. One time, with death from the patient's disease forty-eight hours later. Perhaps it was an error to have explored so ill a patient.

It is our practice to get patients in whom exploration has been done out of bed early after operation, as they easily become bedfast and die.

Palliative Operation. Palliative operation was done in 20 patients, or 30 per cent of the whole number seen, or 48 per cent of those subjected to operation. The operations were as follows:

Gastrojejunostomy 16 (15 posterior, 1 anterior); for pyloric obstruction.

Gastrogastrostomy, 1 for hour-glass obstruction.

Gastrostomy, 3, for cardiac obstruction.

TABLE 3.—OPERATIVE MORTALITY OF VARIOUS TYPES OF OPERATION FOR CANCER OF THE STOMACH.

Author (See Table 1 for deaths.)	Exploratory deaths.		Palliative deaths.		Resection deaths	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Alexinski	..	.	..	...	148	50
Altschul	19	13	54	27	20	40
Bagozzi	5	20	8	25	14	42
Bastianelli, P.	9	11	22	23	21	21
Bastianelli, R.	..	15	..	21	...	28
Billroth	..	..	14	50	16	56
Bindseil	..	...	..	.	8	21
Borelius	21	16	38	16	31	21
Brunner	8	26				
Carle and Fantino	..	...	9	37		
Caspersohn	..	..	..	..	11	55
Cheever	5	20	7	13	3	13
Creite	..	...	..	..	19	38
Crossan	7	41	10	50	5	36
Dancel	4	15	33	16	21	28
Deaver	..	...	..	..	12	26
De Buele	..	...	..	15	...	31
Delore and Alamartine	..	...	..	33	14	33
Delore and Santy	..	...	..	..	1	5
Derjushinsky	..	...	..	..	39	67
Eiselsberg	..	...	13	28	7	31
Faroy	6	...	8	..	7	45
Feurer	..	...	..	..	11	19
Finsterer	..	...	..	..	...	19
Galpern	..	...	..	12	...	12
Gamberini	6	20	6	15	2	7
Gar	..	...	..	..	5	28
v. Hacker	..	...	25	50	19	45
Hanssen	9	16	16	21	5	7
Hitzenberger and Merkler	..	...	50	..	20	
Hoffmann	27	54	..	..	49	50
Kleinschmidt	..	...	12	26		
Küttner	..	...	..	..	25	24
Makkas	..	...	..	..	57	34
Mayo, C.	22	2.9	67	11.1	...	13.7
Mikaye	..	...	..	..	3	11
Nyström	..	...	..	..	46	
Oberniedermayr and Stahnke	..	19%	mortal.	of all operations		
Persson	..	17	..	23	101	27
Petersen	..	...	..	..	...	40
Ribeira and Sans	..	...	24	50	14	48
Schönbauer and Orator	10	9	..	10	33	21
Segre	..	...	4	20		
Serafini	1	3	10	31		
Sherren	..	...	..	..	3	11
Steudel	..	...	16	35		
Stich	..	...	19	34	7	26
Stumpf	..	...	..	..	2	9
Taniguchi	..	...	..	..	140	25
Témoin	..	...	..	..	100	15.5
Vecchi	1	4	10	18	6	24
Volkmann	..	...	..	..	18	32
Weil	..	...	..	..	31	25
Ashhurst and Klopp	7	44	7	31	1	16.6

The anesthetic used in our palliative operations was:

Ether. Five times, with 1 death from surgical shock eighteen hours after operation.

Nitrous oxide gas, eight times, with 3 deaths; 1 after forty-eight hours, from the patient's disease; 1 after seventeen days, from multiple pulmonary abscesses; 1 after one month, from progress of the disease.

Local. Six times, with 3 deaths, all within a few days, from asthenia.

Spinal. One time, with recovery.

The following table shows the reasons why no radical operation could be undertaken in these 20 patients:

PALLATIVE OPERATIONS.

Name and age.	Nature of growth.	Metastasis.	Patient's condition.	Lymph nodes.
1. Louis S., 60	Fixed	Liver and pancreas	Poor	++
2. Charles G., 52	Entire stomach	0	Fair	++
3. Lizzie W., 60	Movable	Liver	Poor	++
4. Jos. A., 64	Fixed	Ascites	Poor	++
5. Jos. B., 55	Movable	0	Very bad[1]	0
6. Paul K., 55	Fixed, cardia	0	Fair	0
7. Louis L., 63	Fixed	Liver	Poor	+
8. Julia S., 45	Fixed	0	Bad	+
9. Samuel E., 55	Fixed	Retroperitoneal	Fair	++
10. Albert L., 58	Movable	Liver and retroperitoneal	Fair	++
11. John W., 77	Fixed	Retroperitoneal	Fair	++
12. Bridget O'D., 55	Extensive but movable at pylorus	None	Fair	++
13. John J., 60	Fixed	Liver and retroperitoneal	Poor	++
14. Peter C., 58	Fixed, cardia	Retroperitoneal	Fair	0
15. Freda B., 60	Fixed	Liver	Fair	++
16. George K.	Fixed	Retroperitoneal	Fair	00
17. Benjamin S., 79	Fixed	Retroperitoneal	Poor	?
18. Herbert K., 46	Fixed, invading colon	Retroperitoneal ascites	Bad	++
19. Mary C., 48	Fixed	Liver and retroperitoneal	Poor	++
20. Gus. S., 58	Fixed	Retroperitoneal	Fair	++

[1] Lost 75 pounds in last ten weeks.

MORTALITY OF PALLIATIVE OPERATION. Our general mortality of palliative operation (35 per cent) is very high. (See Fig. 7; Table 3.) Of the 13 patients who survived, the subsequent history is known in 11. These patients were discharged from the hospital

and *lived in comfort* for periods varying from three to fourteen months, before being incapacitated by their final illness, which usually lasted only a few weeks. The patient who had the shortest period of relief after operation (three months), curiously enough, had the longest period of disability (four months) before death. The patient with the longest period of good health (fourteen months), was a man of seventy-seven years at the time of operation; fourteen months later he died suddenly, of *angina pectoris*, having had entire freedom from gastric symptoms since operation.

We believe that palliative operations are worth doing in spite of the high early mortality. As Abernethy said of malignant disease in general, "To forbear to operate is to consign the patient to hopeless misery."

Radical Operations. Only in 6 patients (less than 11 per cent of those treated) was it possible to attempt radical operation (Fig. 4). This small percentage is not due to our selection only of the easiest cases, for partial gastrectomy was attempted in every case in which it was thought the patient would survive.

Mortality of Partial Gastrectomy. One of the 6 died soon; a second died, probably of lung metastasis, four and a half months later; and 4 are still (April, 1932) living, from seven to thirteen years after operation. (Fig. 7; Table 3.) A summary of the gastrectomy case records follows:

1. Benjamin, E., aged forty-two years. Admitted August 2, 1919; discharged October 20, 1919. Well thirteen years.

Notes in 1919: Following influenza in 1918, has never been strong enough to work. Normal weight was 160 pounds. In the three months before admission it dropped from 144 to 125 pounds. His chief complaint was weakness and loss of appetite; heaviness after eating, relieved by self-induced vomiting. For two months before admission, he had symptoms of pyloric obstruction. *X*-ray showed retention.

Operation. August 8, 1919 (ether), disclosed a movable mass at the pylorus with enlarged lymph nodes in gastrohepatic and gastrocolic omenta. No metastasis to liver. Partial gastrectomy.*

Patient gained 25 pounds in three months following operation. Present weight (1932) about 157 pounds; no complaints at all. Works daily as a clothing finisher. Appetite normal; no digestive disturbances except a tendency to constipation, relieved by an occasional dose of laxative. Abdominal scar firm.

* Polya technique. Convalescence delayed by bad infection of abdominal wound attributed to neglect of operators to change gloves after completing the gastrectomy. Secondary suture of incision, November 21, 1919.

Laboratory Reports. Large callous ulcer involving nearly the entire circumference of the stomach.

Histology. Adenocarcinoma. *Lymph nodes* in gastrohepatic and gastrocolic omenta show carcinoma. (Patient was presented October 28, 1925, at the Philadelphia meeting of the Clinical Congress of Surgeons, clinic at the Episcopal Hospital.)

2. Ernest M., aged fifty-five years, referred by Dr. H. M. Freas, admitted September 28, 1920; discharged October 23, 1920. Well twelve years.

Indigestion and belching for eighteen months, vomiting for nine months; lately coffee-ground vomitus; 50 pounds loss of weight. *X*-ray showed retention of one-quarter of the barium meal after six hours; a mass, probably operable, at the pylorus, with crater of original ulcer, and larger surrounding mass of carcinoma.

Operation. October 1, 1920 (gas and 90 cc. ether), disclosed a mass the size of an ordinary apple in the prepyloric region of the stomach, adherent to the pancreas, causing hour-glass deformity. No metastasis to liver. Partial gastrectomy (Polya); difficult dissection of stomach from pancreas. Uneventful recovery.

Gained 35 pounds within one year after operation. Twelve years later, at the age of sixty-seven years, weight is normal (145 pounds); has no digestive disturbances, but eats smaller meals and at shorter intervals than before operation. Occasional laxative necessary. Works daily as a tooldresser.

Laboratory Reports. Chronic perforation of stomach at lesser curvature on posterior wall. Independent and contiguous is a carcinomatous ulcer about 8 cm. in diameter, occupying the entire remaining lumen of the stomach. *Histology.* Colloid carcinoma of stomach. (Patient was presented October 28, 1921, and October 28, 1925, at meetings of the Clinical Congress of Surgeons in Philadelphia, clinic at the Episcopal Hospital.)

3. Julia E., aged sixty-five years, admitted September 25, 1921; death September 29, 1921, forty-eight hours after operation.

Complained of vomiting, nausea and the loss in weight of 34 pounds during five or six months. Lately has had frequent vomiting of coffee-ground material. *X*-ray was not taken.

Operation. September 27, 1921(gas), disclosed a tumor in the body of the stomach, from one curvature to the other, with enlarged lymph nodes in the gastrohepatic and gastrocolic omenta. No metastasis found in liver. Subtotal gastrectomy (Polya). Had to be catheterized three times after operation and had gastric lavage three times for vomiting. Death, forty-eight hours after operation from asthenia. Necropsy showed anastomosis intact, no peritonitis; metastasis in right lobe of liver, not disclosed at operation.

Laboratory Reports. On lesser curvature, an ulcer 7 cm. in diameter, circular, with indurated raised walls; cardiac section only 1.5 to 2 cm. from ulcer; pyloric section 5 cm. from ulcer. *Histology.* Medullary carcinoma.

4. Mary T., seventy-five years, referred by Dr. R. S. Hooker; admitted April 12, 1924; discharged April 30, 1924. Death, four and a half months after operation.

For eight weeks preceding admission had epigastric pain; and for the past two or three weeks, vomiting. Normal weight 145 pounds, but lost 43 pounds in eight weeks. *X*-ray showed retention of three-fourths of the opaque meal after six hours and a filling defect at the pylorus.

Operation. April 15, 1924 (gas), disclosed a mass at the pylorus, but no metastasis to liver. Partial gastrectomy (Polya). Uneventful recovery.

Was comfortable until a few weeks before death (August 27, 1924), four and a half months after operation, after an illness characterized by cough and expectoration. (Probably pulmonary metastasis.) No necropsy.

Laboratory Reports. Specimen measures 12 cm. on lesser curvature, 15 cm. on greater curvature; duodenal end passes 2 or 3 cm. beyond the gross lesion, and the cardiac 5 or 6 cm. *Histology.* Adenocarcinoma; lymph nodes in both gastrohepatic and gastrocolic omenta show early carcinoma; pyloric cut end of specimen shows carcinoma: cardiac cut end of specimen shows no carcinoma.

5. Harry W., aged sixty years, admitted January 7, 1925; discharged March 6, 1925. Well seven years.

Chief complaint, pain after eating for five months previous to admission, relieved by vomiting. Twenty-three pounds loss of weight. *X*-ray showed retention of one-sixth of the barium meal after six hours; filling defect in the prepyloric region, probably from adhesions; if a carcinoma, probably small and operable.

Operation. January 19, 1925 (gas), disclosed a freely movable mass at the pylorus; no metastasis to liver; enlarged lymph nodes in the gastrohepatic and gastrocolic omenta. Partial gastrectomy (Polya). Recovery complicated by persistent hiccough from January 23 to 31, 1925. One stitch abscess.

In good health in April, 1932, seven years after operation.

Laboratory Reports. Operative specimen measures 11 cm. on the lesser curvature, 16 cm. on the greater curvature. There is an annular carcinoma at the pylorus. The pyloric section passes 2 cm. distant from the gross lesion, but a record of the cardiac end is lost.

Histology. Adenocarcinoma. The cardiac cut-end shows freedom from carcinoma; gastrocolic omentum lymph nodes show carcinoma. One lymph node from the gastrohepatic omentum shows carcinoma.

6. John D., aged sixty-eight years, admitted February 10, 1925; discharged March 16, 1925. Well seven years.

For six months had constant gnawing pain in epigastrium, beginning one or two hours after meals, sometimes relieved by eating. Lately had belching and small amounts of vomitus and lost 25 pounds. *X*-ray showed filling defect at pylorus, but no obstruction.

Operation. February 11, 1925 (gas) disclosed a movable mass at pylorus; no metastasis to liver; lymph nodes beneath pylorus and in gastrocolic omentum enlarged. Patrial gastrectomy (Polya). Uneventful recovery. Seven years later, in April, 1932, aged seventy-five years, well, except for feebleness of age. *X*-ray (January, 1930) shows no carcinoma in the stomach; no obstruction at anastomosis and a shadow in the lung suspected of being due to metastasis. Clinically, there were no symptoms nor physical signs of pulmonary lesion as late as April 15, 1932.

Laboratory Reports. Specimen measures 10.5 cm. on lesser curvature; 18.5 cm. on the greater. *Histology.* Adenocarcinoma; both cardiac and pyloric cut ends of the specimen show carcinoma.*

Lymph nodes in both gastrohepatic and gastrocolic omentum show carcinoma.

* I have reviewed the slides of this specimen, and I can find none which show this. The slides found show, in my opinion, freedom of carcinoma or any question of carcinoma at the cardiac and pyloric sections. (J. W. K.)

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CANCER: AS A PROBLEM OF THE BIOLOGY OF CELL DIVISION.*

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The successive complete expression of the cellular properties of proliferation, differentiation and organization is essential for the development of a normal, functionally effective organism or part thereof.

The partial or complete lack of expression of any one of these three basic biological growth processes results in disharmony and inadequacy or injury to the well-being of the individual in which this occurs.

Cancerous growths are examples of such inharmonious and incomplete developments. Even though it is true that neoplasms are primarily expressions of misplaced proliferation activity, it is of equal biological significance that cellular differentiation in these growths is scanty, while organization to functional utility is entirely absent.

From the developmental point of view it is clear that the breakdown has not occurred in the process of proliferation, but in that of the successive stages of differentiation and organization increasingly.

Now since cellular differentiation and organization are successive steps following proliferation in normal development, and since these are unexpressed in fulness in malignant neoplasms, and since the basic universal characteristic of cancer is cellular proliferation, it is obvious that an understanding of the mechanism of this disease from the point of view of development is best first attacked through a study of the chemistry of cell proliferation.

This has been the central problem of the work at our Institute for the past four years. From it the following facts have evolved:

The simple chemical group sulfhydryl is present in high concentrations where cells are dividing. When the group is removed either experimentally or by the natural course of the oxidative and hydro-

* Read May 4, 1932.

lytic metabolism of growth, cell division stops. When the group is presented in increased amount to incompletely differentiated cells capable of division, the percentage rate of multiplication is increased.

This response occurs in organisms and parts thereof, from the lowest to the highest. From plants, through protozoa, molluscs, crustaceæ and rodents, up to man, is this growth-accelerating group effective.

All types of expression of growth by increase in cell number are stimulated by this naturally occurring chemical group. Reproductive, regenerative, repair, replacement, embryonic, phylogenetic, normal and pathological cell proliferations respond in uniform manner.

From all of this it follows that sulfhydryl is the naturally occurring, essential and specific, chemical stimulus to cell division.

By its proper exhibition to growing things, such as the skin of the mouse and rat, the aberrant growths resulting from injury to the regenerating chela of the hermit crab, and the growth center of the distal segment of this same structure, it is possible to produce wild and unorganized overgrowths. These, in their architecture and cytology, resemble the picture given by malignant growths in man. Because of these facts, and the fact that the growth of malignant transplantable tumors of rodents is accelerated by this chemical group, it is clear that sulfhydryl is the basic naturally occurring chemical group fundamentally responsible for the proliferation phase of cancer growth, as it is for normal.

But this is not the whole story. Cell proliferation as a phase of development is not a continuous process. Sooner or later this ceases and the only divisions which normally occur are those which give rise to cells replacing others which have been lost or destroyed by use or injury. That is to say, the cell proliferation of development normally becomes equilibrated.

Now it can be stated that the basic laws of chemistry are just as effective in living things as they are in the test-tube, since the mechanics of living are but the expressions of underlying chemical reactions.

This being so, and the fact having been established that sulfhydryl is the key group for cell proliferation, it follows that sulfhydryl is the key group of a chemical equilibrium through which growth by increase in cell number is regulated.

Taking the fact that the sulfur of living protoplasm is present in the reduced state, and the fact that the sulfur as excreted in useless form is completely oxidized, it follows from what has gone before that just as sulfhydryl is the essential specific chemical group stimulative of cell proliferation, so should it naturally occurring partially oxidized derivatives be inhibitive of this process. And our experiments have shown that this prediction based on the known laws of chemistry is true, for every species and type of growth expression noted in the preceding paragraphs.

The postulate can therefore be considered as having been established that the equilibrium of growth by increase in cell number is regulated by the naturally occurring chemical equilibrium the components of which are the sulfhydryl group and its partially oxidized derivatives.

Just as this applies to normal proliferation growth, so does it apply to malignant. For partially oxidized sulfhydryl not only retards the growth of transplantable and spontaneous mouse tumors but also prevents the proliferative development of aberrant, unorganized growths normally eventuating from injury to the regenerating chela of the hermit crab.

This is as far as we have gone with certainty. Wherein lies the basis of the breakdown observed in the cellular differentiation and organization of cancer has yet to be ascertained. The next and infinitely more difficult step is a determination of the chemical factors for differentiation and organization in normal development, and the demonstration that their availability or utilization is defective in malignant growth.

Our work along this line, so far, has shown that in the absence of injury or a specifically sensitive growth center acceleration of proliferation causes orderly enhancement of differentiation and organization, while retardation of proliferation results in an orderly holding back of these developmental processes.

From this it follows that increased proliferation *per se* is not the cause of the inadequate differentiation and organization exhibited in malignant growth. The basis of this is undoubtedly a distortion of cell chemistry from its normal path, which is either constitutionally determined or induced by extraneous factors.

THE IMPORTANCE OF THE DIFFERENTIAL DIAGNOSIS OF TUMORS*

By JOHN H. GIBBON, M.D.

The title of this contribution is not exactly that suggested by the Committee, which was "The Recognition of Cancer with Special Reference to the Untrustworthiness of Symptoms," but I think the revised title is comprehensive enough to include a presentation of the very important idea of the original one, and also admits the introduction of certain phases of the subject which to my mind need particular emphasis.

One of the accomplishments of those, both medical men and laymen, who have interested themselves in the "cancer problem" is that the laity pretty generally have been impressed with the importance of having examined any "lump" or excrescence which they may detect. These cases do not offer much diagnostic difficulty. Also, as the result of propaganda, patients are paying more attention to symptoms and many are presenting themselves for examination because of the fear of cancer, and it is this group which demands the exercise of every diagnostic measure, of skill and of judgment. Among these will always be found a small percentage of cancers, a larger percentage of benign tumors and other pathological conditions and a still larger percentage suffering only from cancerphobia. These patients who are the victims of a variety of lesions or of fear alone, and who come to us largely as the result of our own general solicitation, deserve skilful examination and conscientious advice. An error in diagnosis represents a failure on our part to carry out a compact. The examiner who does not feel himself capable of making a diagnosis should never fall into the error of making light of symptoms and giving unwarranted assurance of health, nor should he fall into the equally unjustifiable but much more common error of prescribing treatment. Many of these patients, who have not been given a thorough and complete examination, are given false assurance, are carried on with symptomatic treatments

* Read May 4, 1932.

or are subjected to unnecessary operation or radiation. The diagnosis then becomes the most important aspect of this whole subject, if mistakes are to be avoided.

Many patients who have been impressed by reading "cancer literature" show a lamentable judgment in the selection of "a doctor," going too often to the man who has a treatment, but no diagnostic ability and often no conscience. The universal fear of cancer has been a potent reason for the development of numerous "cancer cures," which often include an impressive but perfectly inadequate examination. The examination is free, but the "cure" demands an outrageous price paid in advance. The ingenuity and refinement of the knavery which produces these fake cures is shown by their pseudoscientific literature which greatly impresses the uninitiated, particularly those suffering from cancerphobia.

Perhaps an example of the working of one of these agencies may better indicate their wickedness, though to most of you it may be altogether unnecessary.

A woman, aged sixty years, is referred by her physician to a surgeon because a cancer of the tongue is feared. A careful examination showed nothing whatever to suggest cancer or any other lesion of the tongue. (The large vascular papillæ at the base are frequently the cause of careful self-examination by the cancerphobiac.) When assured that she had no cancer and charged an ordinary office fee, she broke into tears and said that but for the lack of money she would be undergoing a course of treatment for cancer of the tongue. She had had a free examination by a representative of one of these nationally, but discretely advertized, cancer cures, a man who adds to the privileges of the regular medical degree the accomplishments of the osteopath, and told she had cancer and needed, of course, this treatment which would be divided into three parts for which she must pay $200 each in advance. But for her lack of funds, she might have had the distinction of being added to a long list of those cured of what they did not have. The cancer quack's best victim is the *malade imaginaire* and his method is quite like that of the *agent provocateur* or the modern prohibition officer, who incites to crime in order to make an arrest. The charlatan creates a fake disease, applies a fake remedy and obtains a fake cure. The only thing about the transaction which is not a fake is the fee. I am quite prepared to say that anyone who demands a large fixed fee in advance for the treatment of

cancer at once casts suspicion on the treatment as well as on himself, and this accusation particularly applies where the patient is the victim of an evidently far-advanced and incurable cancer.

But let us leave this particularly pathetic side of the picture and go back to the question of diagnosis where errors may be made by the most conscientious, especially if he does not employ every means available.

In the first place I should like to say that it is quite possible for the most apparent cancerphobiac to have cancer, as often there may be a real cause for this fear. Nothing constitutes a sadder commentary on one's ability than to reassure one of these patients and then have him turn up later with cancer. Careful and complete examination, often repeated at intervals, is the only way to avoid this lamentable error.

There are certain regions of the body where cancer develops insidiously and may avoid detection by the ordinary examination until it has reached a far-advanced stage. The large intestine, particularly the rectum, is one of these regions. An experienced surgeon of a generation ago told me the large majority of cases of cancer of the rectum he saw had been previously treated (not for what they had), but had not had even a digital examination made, and that about one-half of them had had recent operations for hemorrhoids. Probably no surgeon could say the same today, but it is still too often true that the lesion in the rectum and distal colon remains undiagnosed, in spite of suggestive symptoms, until a stage is reached when palliation alone is applicable. This is to be regretted because cancer of the larger intestine does not metastasize early, except in the young, and the results from resection are as good, if not better, than those obtained from similar treatment of cancer in other organs. Fifty per cent of five-year cures may be expected in these cases if operated upon before metastasis has occurred. In a recent publication[1] we reported 17 resections of the colon for cancer. In this group there were 5 operative deaths. Of the 12 patients surviving operation, 3 died subsequently of cancer, but each had metastasis at the time of operation. One has a recurrence who did not have metastasis when operated upon and 8 remained well from eight years to eighteen months after operation.

In the diagnosis of lesions of the rectum and lower sigmoid too

[1] Ann. of Surgery, 1932, **96**, No. 1.

much reliance has been placed on the results of roentgen-ray examination which are often negative in the presence of a growth. No examination is complete without a careful proctoscopic inspection. In the colon above the rectosigmoid the roentgen-ray examination, provided it includes a barium enema, is our most reliable diagnostic means. Alteration in the habitual function of the colon and rectum constitutes the clinical indication for further examination and the absence of blood or mucus in the stools is not sufficient cause for delay of the visual and roentgen-ray examinations.

Early diagnosis of cancer of the stomach is usually very difficult because the disease develops insidiously and too often gives no more than a suggestion of a gastric lesion. There is, as a rule, a marked contrast between the symptoms of a gastric or duodenal ulcer, which are generally quite characteristic and of great diagnostic value, and the entire absence of characteristic symptoms in cancer of the stomach. In the latter case much more valuable than symptoms are the results of blood examination, gastric analysis and gastrointestinal roentgen-ray study. No cancer of the stomach is apt to escape detection at the hands of a good roentgenologist, and from no other source can such valuable information be obtained. No treatment then is justifiable in the presence of gastric symptoms or of symptoms which may be caused by a gastric lesion until these examinations have been made. This caution is particularly important because cancer of the stomach runs a rapid course and valuable time is lost in giving treatment without a complete examination.

The breast, a common site for the development of cancer, is also an organ in which benign lesions are frequent and a differentiation between the malignant and benign tumors becomes at once important if we are to save life in the one case and avoid a needless sacrifice and disfigurement in the other. There was a time when some surgeons thought that the presence of any noninflammatory mass in a breast constituted a justification, if not an indication, for radical amputation. Such an attitude seems to me indicates a regreattable lack of diagnostic ability and surgical judgment. On two occasions I have grouped a fairly large number of breast operations, and in about 45 per cent the operations were limited and nondisfiguring and done for benign tumors and cysts. A radical operation in this large percentage of patients would have represented a woeful lack of surgical judgment. In nearly every case of breast tumor a sufficiently accurate diagnosis of the type of the

tumor can be made on history, symptoms and physical characteristics to guide one in determining the kind of operation indicated. Where doubt exists the complete removal of the tumor itself and its macroscopic examination will be enough to decide the question of the necessity for the radical operation. Incomplete removal for biopsy in breast tumors is to be condemned. It seems to me that the surgeon should determine in nearly every case the extent of the operation when the doubtful tumor has been inspected and that to wait for a histological examination is in most instances a mistake. I have not great confidence in the examination of frozen sections unless it confirms the clinical history, physical character and macroscopic appearance. Here again it becomes a matter of importance that the examiner should know the characteristics of the different types of breast tumor and be able to differentiate between them. Most examinations of the breast are too perfunctory and stop on the determination that there is or is not a tumor. This cannot be excused on the ground that benign tumors and cysts are apt to become malignant and, therefore, should be treated as such, for this is not the case. The benign tumors are not apt to undergo a malignant change nor are the cysts with the exception of the papillary cysts or tumors which develop usually in the center of the breast, under the areola, and which are often accompanied by the discharge of dark blood from the nipple. A serous or slightly sanguinous discharge from the nipple is not a sign of malignancy, often occurs after cessation of menstruation and, if unaccompanied by a cyst, usually spontaneously stops. Bleeding from the nipple in cancer, with the exception already mentioned, is a late symptom and one that is not necessary for a diagnosis. No patient with a benign tumor or cyst or with pain or discomfort in the breast should be told to "forget it," but in the one case the tumor or cyst should be removed, in the other reëxamination at regular intervals should be made. In examination of the breast the patient should be examined in both the recumbent and sitting position. Often tumors in the breast and glands in the axilla may be found in the one position which escape detection in the other. The palpation of the breast between the fingers and thumbs is the poorest way of finding a tumor. The best way is by palpation with the open hand against the chest wall with the patient recumbent. The examination of both breasts is most important.

One might continue to discuss cancer in the other organs of the

body and show further reasons for careful study and diagnosis. For instance, malignant change in the adenomatous goiter is much more common than used to be thought, amounting to at least 3 per cent. Therefore, we can no longer consider the deformity and the possibility of a toxemia as the only reasons for operation. Enough, however, has been said to indicate that if we as clinicians are going to add anything to the advancement of this subject it will probably be through a more careful study of the individual patient, the employment of all means available for diagnosis and by increasing our knowledge of the life history and characteristics of the different types of tumor.

In conclusion I would suggest that the patient having a tumor or the fear of one needs to have impressed upon him the importance of diagnosis and the selection of some one capable of making it and he should not exercise himself over the choice of treatment until every diagnostic means has been conscientiously employed.

THE TUMOR CLINIC*

By BURTON T. SIMPSON, M.D.

(Director, New York State Institute for the Study of Malignant Diseases, Buffalo, N. Y.)

I APPRECIATE the honor of being asked to take part in this symposium on cancer, and I wish to express the pleasure I had in listening to the papers that preceded mine. The Society is to be congratulated on the character of these papers and the valuable methods brought before you. The subject I have been asked to talk upon is "the tumor clinic."

At the meeting of the American College of Surgeons held in Chicago on October 17, 1929, the Board of Regents voted to undertake the promotion of better cancer service throughout the continent and entrusted to the committee on the treatment of malignant diseases the task of perfecting the details by which this could be accomplished.

This committee was composed of surgeons, radiologists and pathologists. Dr. Pancoast was a member of the committee. In its survey the committee reported the facilities for cancer control under the following heads:

1. Cancer institutes.
2. Cancer research laboratories.
3. Cancer hospitals.
4. Cancer clinics in general hospitals.

In 1930 the clinical congress of the American College of Surgeons, which met in Philadelphia, held a symposium on the cancer facilities throughout the United States, based on a report of the committee for the treatment of malignant diseases. Dr. James Ewing, who spoke on the subject of the "cancer institute," astounded the audience by making the statement that a properly equipped cancer institute should have an endowment of $10,000,000 and that several of these should be established at strategical points through-

* Read May 4, 1932.

out the United States. Unfortunately, at this time we do not have any institute of this magnitude.

The State Institute at Buffalo, with which I am associated, has an investment of $1,000,000 and an annual appropriation which averages $200,000. The Memorial Hospital in New York, which should now be considered as a cancer institute, has an annual income in the neighborhood of $400,000, of which about $100,000 is in the nature of endowments and donations.

The cancer institute is, of course, the ideal to be desired for it attacks the cancer problem from all angles and deals not only with the treatment of cancer but carries on considerable research.

The cancer hospital is established primarily for the treatment of cancer cases. The men attached to such institutions are specialists in their various lines and devote their entire time to cancer therapeutics. It is needless to say that such an institution should be fully equipped with the modern facilities for the treatment of this disease.

The subject which we will deal with especially tonight is the tumor clinic in connection with the general hospital. However, I believe every populous area should have a cancer center with a cancer institute or a cancer hospital or both which could act as a clearing house for its locality and could also carry on experimental research.

Before I take up the subject of the tumor clinic in the general hospital I should like to review briefly what New York State is doing in the question of cancer control. New York State is commonly divided into the up-state district and the Metropolitan area and I shall use this division in speaking about the facilities.

Up-state. In the up-state district we have the cancer institute located at Buffalo. This institute was created to study cancer from the laboratory standpoint and was the conception of Dr. Roswell Park, professor of surgery at the University of Buffalo. In 1898 the State Legislature appropriated $10,000 for this purpose and founded the first cancer laboratory for the exclusive study of this disease in the world. In 1910 the name was changed to the State Institute for the Study of Malignant Diseases under a Board of Trustees and the activities of the Institute were increased to include the treatment of cancer patients as well as to carry on experimental research. At the present time the Institute is composed of the Gratwick Laboratory, which is devoted almost entirely

to research; the hospital building, for the housing of cancer patients; the clinic building, for examination of patients and treatment with radium and *x*-ray; and the biological station at Springville, where much of the animal experimentation is carried on. The Institute has all the modern facilities for research work and the treatment of cancer. It is composed of a pathological department, a biochemical department, a physical department, a biological department and a therapeutic department. The head of each department is a man highly qualified in his line of work. The fundamental object of the Institute is research. However, the pathological laboratory makes free diagnoses of pathological tissues for any physician in the state, and the hospital treats any patient sent to the Institute free of charge. The Institute has nearly 8 grams of radium of which 2 are in solution and from which emanation is pumped daily, 4 grams are utilized as a radium pack and the remainder is distributed in tubes, plaques and needles. At the present time the therapeutic department is bending its greatest energy along the lines of the treatment of malignant disease by radiation. However, all forms of cancer treatment are carried on in the hospital.

There are in the up-state area complete tumor clinics in connection with the Buffalo City Hospital, the Strong Memorial Hospital at Rochester and the Memorial Hospital at Albany, New York. Several of the smaller cities have diagnostic tumor clinics in connection with their hospitals.

Metropolitan District. Greater New York is well equipped to handle the cancer problem. There has been established under the Department of Hospitals a division of cancer which comprises: (1) the radiotherapeutic department which is located at Bellevue Hospital and where most of the radiotherapy is carried out; (2) at Fifty-ninth Street is the cancer clinic, which serves as a diagnostic clinic, although treatments by radiation and surgery are also given at this clinic; (3) the Custodial Hospital on Welfare Island, which is devoted largely to the care of advanced cancer cases (however, radiological and surgical treatment is given here to many of the patients; there is also an emanation plant at the Custodial Hospital which supplies the radon for the entire division); (4) the Cumberland Hospital in Brooklyn is also a part of the unit, and is in the nature of a cancer hospital. The New York City Division of Cancer has more than 6 grams of radium and all the other adequate facilities for the diagnosis and treatment of malignant disease.

The Memorial Hospital in New York may be considered today as one of the ideal cancer institutes. Besides the large number of cases of cancer being treated at this hospital by modern methods, much of the best research in the therapeutics of cancer done in this country emanates from this institution.

The following hospitals of New York City have complete tumor clinics in connection with their other services: Montefiore, Stuyvesant Square, St. Luke's, Women's, Beth Israel, Presbyterian and Mount Sinai.

In our discussion tonight we are especially interested in the question of tumor clinics in general hospitals. These may be of two varieties, viz., (1) a diagnostic clinic and (2) the complete tumor clinic.

Because of the fact that cancer now ranks second as the cause of all deaths and also that it is apparently on the increase, it is realized that every effort must be made to combat this disease and it has been recognized that the general hospitals have a responsibility in this matter. It has therefore been urged that the general hospital organize a tumor clinic to assure the best interest of the cancer patient in its community. This problem has been recognized by the American College of Surgeons which is at present using its influence and facilities to organize tumor clinics in general hospitals. The problem has also been recognized abroad and many excellent cancer centers have been established, especially in France, England and Germany. The idea of the tumor clinic in general hospitals has the support of most of the leading men interested in the cancer problem in this country. It is now recognized that the word "cancer" covers a group of diseases and that the advances in the field of diagnosis and treatment of this group of diseases have been such that it is difficult for any one man to cover the entire field. Therefore, the organization of a group of men from the staff of the hospital who are especially interested in the cancer problem and the pooling of the facilities into one department appears to be the best method of handling the cancer question at the present time.

The organization of a tumor clinic offers numerous advantages among which may be mentioned a center for diagnosis, the proper care for the patient, lay and professional education, modern treatment facilities and a teaching medium for both students and professional men.

Organization. Two types of organization of clinics in connection with general hospitals present themselves for consideration:

1. *A Cancer Division.* This is to be headed by a director who is a full-time man and is trained and skilled in surgical pathology, cancer diagnosis, cancer surgery and radiation therapy. All cancer cases that come to the hospital should be referred to the director and he should pass upon the diagnosis and suggest the treatment. The ward cases should be treated either by him or under his direction. The private cases should have the advantage of his advice and, when necessary, his assistance in applying radiation. The director should be available for consultation to any member of the staff without fee. To my knowledge there are but three hospitals in this country which are functioning with this arrangement.

2. *Group Cancer Clinic.* This is the type that is available to all general hospitals and is the kind which is recommended by the American College of Surgeons.

Staff. In the organization of the group clinic the personnel of the staff is of great importance. Fundamentally this should be composed of a surgeon, a radiologist and a pathologist. This fundamental group should be supplemented by other men available on the hospital staff. The following specialists are desirable—a gynecologist, a genito-urinary surgeon, a dermatologist, a laryngologist, an internist, a proctologist, a dentist or any other specialist who is interested in the cancer problem and who is available. It is important that the chief of the staff have special training and ability in the cancer field and that he is enthusiastic about the cancer clinic.

Diagnostic Facilities. It is important to have in the group a tumor pathologist. By that I mean a pathologist who is competent to properly diagnose, histologically, tumor tissues. It is essential that biopsy be done in many cases and facilities for frozen section for immediate diagnosis be at hand. *X*-ray facilities for fluoroscopy and diagnostic films are important in connection with the tumor clinic. The facilities for endoscopy are necessary and can be well carried out by those specialists who are skilled in the use of the different types of endoscopes.

Therapeutic Facilities. It is not necessary to speak of surgery as every general hospital has excellent surgeons and surgical facilities. However, these should be available for the tumor clinic and should include diathermy and electric cautery. A high-power

x-ray machine of 200 k.v. should be available. The minimum amount of radium salt that should be accessible is 200 mg. However, more radium would be preferable and when possible an emanation plant is very valuable. However, at the present, it is possible to obtain radon from chemical companies.

Record System. A uniform system of records is very important and there is now available a series of uniform records which are recommended by the American College of Surgeons for use in tumor clinics. It is needless to say that a record clerk is necessary.

Social Service. It is very important that a social service be conducted in connection with the tumor clinic for it is essential that patients be returned for reëxamination and that the records be properly completed.

Clinic days should be held weekly, semiweekly or daily, according to the size of the hospital and the number of tumor cases in the vicinity. On these days the members of the staff should be present to diagnose new cases and to reëxamine patients. All members of the staff of the hospital should be encouraged to refer their tumor cases to the tumor clinic and all physicians in the neighborhood invited to bring their cases there for diagnosis and advice.

It is essential to have monthly staff meetings at which time there should be a general discussion of the tumor cases that have been seen at the clinics and all matters that pertain to the welfare of the organization be brought before these staff meetings.

No definite rules can be formulated for the conduct of these clinics. Each staff must make its own rules and regulations in regard to referring cancer cases to the clinic. The success of these clinics depends, of course, on the enthusiasm and coöperation of the members of the staff and their conscientious attendance at all clinics.

It is my belief that the establishment of a tumor clinic in at least one general hospital in the community will be of great value to the individuals in that community and will be a great step forward in the solution of the cancer problem.

A valuable summary of the question of the tumor clinic was made by Dr. Greenough in his article on "Tumor Clinics," and in closing I would like to quote to you his summary on the advantages it offers.

"1. The work in cancer cases is placed in the hands of those members of the staff who are most interested in that subject.

"2. Opportunity is given by consultation with representatives of

the clinical, pathological and radiotherapeutic departments for mutual instruction and increase of knowledge of the disease and its treatment.

"3. A definite rational plan of treatment can be determined for each individual case, after consultation; and the results can be checked and discussed by the group, in the subsequent follow-up.

"4. Concentration of the hospital material permits greater experience and more productive work.

"5. The standards of the hospital for efficient diagnosis and treatment of cancer are inevitably raised, and this educational effect extends beyond the hospital to medical students and to physicians in general practice in the immediate vicinity.

"6. The additional expense to the hospital involved in the maintenance of a tumor clinic amounts only to about $3000 a year."

CANCER IN ANIMALS*

By HERBERT L. RATCLIFFE, Sc.D.

The present discussion will be limited to those neoplasms of lower animals that may be regarded as analogous to spontaneous tumors of man. Tissue growths occurring under the stimulus of animal parasites, such as sarcomas of the ceca of pheasants infested with heterakis, or carcinomas of the stomach of rodents infested with spiroptera, although often morphologically indistinguishable from true spontaneous neoplasms, will be omitted.

Excluding neoplasms formed under the stimulus of animal parasites, spontaneous cancer occurs in all forms of vertebrate animals, from fish to mammals, and has been reported in invertebrates also. Every type of tumor known for man, with the possible exception of certain growths of brain tissues, has its close analogue in those occurring in lower animals. However, the incidence of neoplasms generally, or any type of neoplasm in all or any animal species, is unknown since, with the possible exception of laboratory rats and mice, adequate surveys are entirely lacking.

Little more is known of the factors favoring development of cancer in lower animals than is known for man. Where sufficient numbers have been studied cancer has been found to occur most frequently in animals approaching senility. Irritants—biological, mechanical or chemical—stimulate neoplasia in susceptible tissues, and hereditary susceptibility to cancer development and to transplantable cancer has been established in inbred or linebred strains of laboratory mice. These findings may, perhaps, apply to all types of animals, but studies have involved relatively few species.

Observations on spontaneous cancer in wild birds and mammals dying at the Philadelphia Zoölogical Gardens over a period of about thirty years shows an incidence of 1.88 per cent for more than 9000 specimens. Comparing the present data (1932) with those compiled by Dr. Fox (1921), when the autopsy records for over 5000

* Read May 4, 1932.

specimens were available for study, it was found that incidence of neoplasms had varied very little for the classes (mammalia and aves) or for the orders of animals represented here. Rodents, carnivora and marsupialia have the highest incidence among the mammals, while the primates, with the largest number of autopsies, have relatively few tumors. Likewise, the birds have shown no pronounced variations: the parrots, psittaciformes, considerably exceed the other orders in frequency of tumor development.

TABLE 1.—SUMMARY OF TUMOR INCIDENCE AND TYPES OF TUMORS COMPARING PRESENT RECORDS WITH THOSE OF 1921

Order.	1921.					1932.				
	Total animals.	Tumor cases.	Per cent.	Tumors.		Total animals.	Tumor cases.	Per cent.	Tumors.	
				E.	F.				E.	F.
Primates, lemures	584	3	0.51	3	0	971	8	0.82	9	1
Carnivora	481	16	3.32	13	5	763	33	4.32	29	7
Perissodactyla, artiodactyla	365	7	1.91	3	4	598*	11	1.84	7	4
Rodentia	198	12	6.06	7	5	431	24	5.56	18	8
Marsupialia	175	7	4.00	7	0	373	9	2.41	8	1
Others	39	3	..	1	2	62	4	..	1	3
All mammals	1860	48	2.58	50		3014	89	2.95	96	
Psittaciformes	689	26	3.77	14	12	1175	42	3.57	24	18
Galliformes	299	1	0.33	1	0	636	9	1.41	7	2
Anseriformes	317	4	1.26	2	2	557	5	0.89	1†	4
Passeriformes	1355	7	0.51	5	2	2837	13	0.46	8	6
Accipitriformes	196	2	..	0	2	315	2	..	0	2
Strigiformes	133	1	..	1	0	311	2	..	2	0
Columbiformes	157	1	..	0	1	280	1	..	0	1
Others	67	2	..	1	1	372	7	..	4	3
All birds	3505	44	1.25	44		6023	81	1.34	82	
All animals	5365	92	1.71	94		9037	170	1.88	178	

* Artiodactyla.
† Three cases not included because of incomplete record.

That there have been no pronounced variations in the incidence of neoplasms in these different groups of animals seems to allow at least two explanations: (1) Susceptibility to neoplasia is equal

in all groups, but unequal adaptability to captivity allows earlier deaths in certain groups, so that there is less chance for tumor appearance, or (2) the variations in incidence indicate natural differences to tumor formation.

Test of either assumption necessitates some basis for age comparisons, since such wide natural difference in length of life, as between the rodents and carnivores, prevent direct comparisons. If, however, longevities of various orders and families be estimated in terms of time required for sexual maturity, comparisons may be made.

TABLE 2.—ESTIMATED SEXUAL MATURITY PERIODS, PROBABLE LONGEVITY, AVERAGE CAPTIVITY PERIOD FOR ALL SPECIMENS AND FOR "TUMOR BEARERS" AND TUMOR INCIDENCE IN THE MAMMALIA

Mammals.	Maturity period (estimated), yrs.	Estimated longevity, yrs.	Maximum captivity period, yrs.	Average captivity period		Tumor, per cent.
				All animals, yrs.	Tumor cases, yrs.	
Primates	2–7	10–50	17	1.5	9.2	0.82
Cercopithecidæ	2–4	15–30	17	1.3	10.3	1.04
Cebidæ	2–4	15–30	13	1.6		
Carnivora	1–4	5–40	18	3.6	9.8	4.32
Felidæ	1–3	8–15	17	3.9	11.5	4.16
Canidæ	1–2	5–10	14	3.5	7.7	5.91
Mustelidæ	1	5–8	14	2.6		
Procyonidæ	1	5–8	15	2.5	5.2	4.00
Viverridæ	1	5–8	14	3.7	5.7	7.55
Ursidæ	2–4	20–40	18	6.5	13.7	6.66
Artiodactyla	2–4	10–30	23	5.3	10.6	1.84
Bovidæ	2–4	10–30	23	4.3	10.7	2.45
Cervidæ	1–2	10–20	19	3.5		
Rodentia	3 mo.–2 yrs.	2–15	18	1.9	4.7	5.57
Muridæ	3 mo.–1 yr.	2–5	7	2.2	3.6	14.92
Sciuridæ	3 mo.–1 yr.	2–5	10	1.5	4.2	4.50
Marsupialia	1–3	5–25	12	2.0	3.3	2.41
Macropodidæ	3	5–25	12	2.1	8.9	1.74
Didelphyidæ	1	5–8	1.3	0.2	0.6	1.54

Limiting discussion to the families because of variation within the orders and comparing estimated longevity to the average captivity periods for the families, it is seen that, in general, when the ratio of estimated longevity to the average captivity period is low the tumor incidence is high. Thus in the muridæ, viverridæ, procyonidæ and canidæ this ratio is about 1 to 3, while in the cercopithecidæ it is about 1 to 12. This is not the whole story, however; three other families warrant attention: the mustelidæ, the

cervidæ and the cebidæ. No tumors were found in these animals, yet in numbers examined and relative longevity they compare favorably with other families in their respective orders.

Another feature brought out in this study is the ratio of the average captivity period for the family to the average captivity period for the "tumor bearers" in that family. This ratio is indirectly proportional to the incidence of neoplasms.

TABLE 3.—MAXIMUM CAPTIVITY PERIODS, AVERAGE CAPTIVITY PERIODS AND TUMOR INCIDENCE IN THE CLASS AVES

Birds.	Maximum captivity periods, yrs.	Average captivity. All animals, yrs.	Average captivity. Tumor bearers, yrs.	Tumor, per cent.
Psittaciformes, psittacidæ	24	3.9	4.7	3.74
Melopsittacus	5	2.1	2.5	15.81
Galliformes, phasianidæ	10	2.1	4.9	1.61
Anseriformes, anatidæ	29	4.8	8.6	0.89
Passeriformes, fringillidæ	10	2.2	4.3	0.68

Comparisons of tumor incidence among the various families of birds represented here is based upon the ratio of average captivity

TABLE 4.—ANATOMICAL DISTRIBUTION AND HISTOGENESIS OF TUMORS

Orders.	Integument.		Digestive.		Genito-urinary.		Respiratory.		Thyroid.		Adrenal.
	E.	F.	E.	F.	E.	F.	E.	F.	E.	F.	
Primates	..	..	5	..	3	..	..	..	..	..	1
Carnivora	4	..	11	..	5	1	5	1	2	1	2
Perissodactyla, artiodactyla	..	..	4	2	2	1					
Rodents	6	2	5	..	5	1	2				
Marsupialia	2	..	4	..	..	..	2				
Others	..	..	..	..	..	2	..	..	..	..	1
Total	14		31		20		10		3		4
Psittaciformes	..	..	3	3	19	1	..	..	..	..	2
Galliformes	..	..	2	..	4	..	1	..	..	..	1
Anseriformes	..	..	1	..	3*						
Passeriformes	..	..	1	..	8						
Others	..	..	5	1	..	1	..	..	..	..	1
Total	..	..	16		33		1				4

E., tumors of epithelial origin; F., tumors of fibrous tissue.
* Not included in total.

periods for all animals to average captivity period for "tumor bearers" in the family. In general when this ratio is low the incidence is high. Differences are by no means so striking as with the mammals, however, and the length of life in captivity is probably not the explanation of the very high incidence of tumors in the undulated grass parrakeets, Melopsittacus undulatus, since they live no longer in captivity than do the fringillidæ (small songbirds).

Tabulation of the tumors in mammals and birds according to their histogenesis and anatomic origin shows that the ratio of tumors of epithelial origin to those of fibrous tissue origin is about the same as that for man.

Considering the classes as a whole, the digestive tract of mammals and the genito-urinary organs of birds are predominant sites of tumor growths. The skin and genito-urinary organs of mammals are also frequently involved, but cancer of the uterus is rare, especially in groups that breed well in captivity, cercopithecidæ, cervidæ and bovidæ.

Cancer of the skin has not occurred in animals exposed to frequent injuries by horns, nor has it been seen in birds.

Comparing these observations to reports of cancer in domesticated animals we find that in the dog, horse and cow the genito-urinary organs are much more frequently involved. However, these records, except for dogs, were obtained from animals slaughtered for food and may not be entirely comparable.

Summary. Neoplasms occur in practically all orders of wild birds and mammals dying in the Philadelphia Zoo. Incidence varies considerably in different groups and seems to be influenced by at least two factors, age and differences in natural susceptibility. Captivity is not thought to have any influences, however. Animals bearing young in captivity have not been subject to tumor growth of the generative organs.

DISCUSSION

Dr. Herbert Fox: I did nothing with the present analysis of the material and can add little to what has been said. I think possibly one feature deserves a little extension. This is the high incidence of neoplasms in the undulated grass parrakeet (Melopsittacus undulatus). The very large number of neoplasms that occur in the adrenal, renal and sex gland group certainly suggests that there must be something there which would

be helpful in the study of neoplasms in general. So far as I have been able to discover there is but a single helpful reference and that is, unfortunately, lost for the time, to the embryology of that organ complex, in parrots. A Russian several years ago studied the histology of the egg in parrots and made the comment that there is a very intricate winding of the tubes that go to make up the primary renal and adrenal tubules in parrots. It may be that in such a work there is something to start studies of tumors that we know develop later in adult form.

I wish to emphasize that in the comparative character of tumors there is something other than simple familial incidence and relation of possibility of life to length of captivity. There is a factor of susceptibility and lack of it. The South American monkey, one of the primates near to man, has a very definite etiological quality. For example, these new world animals have a very much greater resistance to tuberculosis than have the old world Simiidæ and Cercopithecidæ, for which an adequate explanation is lacking. There can very readily be a peculiarity of resistance to tumors on a comparable basis.

OBSERVATIONS ON THE EPIDEMIOLOGY OF POLIOMYELITIS*

By JOHN R. PAUL, M.D.

and (By Invitation)

J. D. TRASK, M.D.

(From the Departments of Medicine and Pediatrics, Yale University School of Medicine, New Haven, Conn.)

In other recent publications dealing with certain clinical and epidemiological aspects of poliomyelitis[1,2] we have pointed out that the term *abortive poliomyelitis* has in the past been loosely applied, although popular and practical usage has tended to designate those cases as abortive, in which signs pointing to meningitic or myelitic involvement existed, but in which no paralysis developed. In other words this concept rested upon the tenet that one could not diagnose clinical poliomyelitis in the absence of signs pointing to meningitic or myelitic involvement. This is quite different from the view originally proposed by Wickman.[3] He felt that the abortive types of poliomyelitis might be manifest by such minor symptoms as fever of short duration, sore throat, headache and vomiting, and that these symptoms might be the *only* manifestations of the disease. In his experience the abortive types were one-third as frequent as the frank cases, but he quoted certain estimates, made during epidemics, in which abortive cases outnumbered the frank cases.† Draper reflects this same concept, intimating that the first phase of the "dromedary" form of clinical poliomyelitis which is often characterized merely by headache, fever, sore throat and listlessness may be the only recognizable phase of the disease in 50 to 80 per cent of all cases of poliomyelitis.[4] Aycock and Luther hold a somewhat similar view but do not attempt to estimate the ratio of what they term suspected abortive to frank cases.[5] The obvious difficulty of subjecting this situation to analysis is that

* Read October 5, 1932.

† We have employed the term *frank case* of poliomyelitis to designate those cases in which tangible clinical signs of meningitic or myelitic involvement occurs.

such nondescript symptoms as those just mentioned do not furnish so-called abortive poliomyelitis with a clinical picture which can be readily identified and that the relationship of the so-called mild or abortive types to frank poliomyelitis has been based on what appeared to be circumstantial evidence. Consequently "abortive" cases have been excluded from most statistics on poliomyelitis. Nevertheless it is needless to point out that, if our concept of poliomyelitis is limited only to the picture presented by the frank case, such a concept will enormously influence views not only on the epidemiology but also on the fundamental nature of this disease.

In our own studies on these problems, which were largely made in New Haven during the summer of 1931, we were immediately confronted with the numerical importance of possible mild attacks of the disease and the problems which they offered in the field of practical clinical medicine and epidemiology. As already stated, these mild or abortive forms owe their identity essentially to the fact that they appear during an epidemic of poliomyelitis and that they affect a fairly similar age group. We have reviewed the clinical symptomatology of a series of 136 examples of these illnesses in another publication.[1] This analysis revealed that, as Draper has pointed out, there is a close similarity between their symptomatology and that of the first phase of the "dromedary" form of clinical poliomyelitis. We believe that careful clinical observations will show that such mild forms of the disease may not be as difficult to recognize as has hitherto been supposed. The most significant observation of this study, however, was the detection of the virus of poliomyelitis in the nasopharynx of 2 children suffering from the abortive form of the disease of the Wickman type. This last finding, which confirms a previous observation made by Taylor and Amoss,[6] furnishes more than circumstantial evidence that there is a common causal relationship between frank poliomyelitis and illnesses corresponding in symptomatology to the first phase of frank poliomyelitis. In other words, we believe that Wickman's original contentions were correct, and we corroborate Draper's views that the majority of individuals who contract poliomyelitis only develop so-called first phase symptoms. Therefore, in spite of the difficulties still inherent in this situation the evidence is sufficient to warrant a re-analysis of the epidemiology of the disease along lines which admit a far broader concept of poliomyelitis than has hitherto obtained.

Our first effort in this re-analysis has been to determine the numerical importance of the so-called abortive types. Data for answering this question have been derived from two sources: (*a*) from a series of 197 poliomyelitis families (that is, families from which a frank case of poliomyelitis had occurred in at least 1 of its members); (*b*) from 3 small communities in each of which 2 or more frank cases had occurred.[7] The family group, exclusive of the initial frank case, gave us a population of 616 individuals under thirty years of age. In this group, secondary cases were recorded in more than one-half of the families and the ratio of secondary frank cases to abortive cases proved to be about 1 to 15. The community group gave us a population of 136 individuals under fifteen years of age and here the ratio of frank to abortive cases was about 1 to 6. As a conservative mean, therefore, we believe that the abortive types may outnumber the frank cases by about 6 or 8 times. This ratio probably differs in different places and in different epidemics but it was about the same in two different epidemics of poliomyelitis which it has been our privilege to observe first hand.* The results imply that the actual attack rate of poliomyelitis is 6 or 8 times that which is usually recorded, and that the disease is highly contagious but fortunately seldom severe.

This concept is diagrammatically expressed in Fig. 1, where the various clinical courses which poliomyelitis may assume is roughly portrayed in terms of a series of hypothetical temperature curves. The distributional percentages of single and dual phase forms of the frank cases have been derived from a series of 195 cases seen in the New Haven Hospital in 1931. The term *minor illness* has been used in this figure to designate abortive forms of the Wickman type.

The value of the recognition of the milder abortive types becomes apparent to the epidemiologist when it is realized that such types may often aid in pointing the way in which the disease spreads either within families or small communities. Within families the listing of abortive as well as frank cases gives important information as to the rate at which the disease spreads through a household. It tends to emphasize the implication that familial cases arise from a common source to which a whole household is not only

* Through the kindness of the staff of the Bryn Mawr Hospital the opportunity has been recently given us to study certain features of the epidemic of poliomyelitis which occurred in Philadelphia during the summer and fall of 1932.

simultaneously exposed, but perhaps heavily exposed because such cases occur within a brief period of one another and are not, as in measles, separated from one another by a period of time which conforms to the usually accepted incubation period.[8] The explanation of this apparent explosiveness of family epidemics is not clear but it should call for renewed efforts to search for the virus in food, milk or water supply, as well as in the throats of individual members of the family.

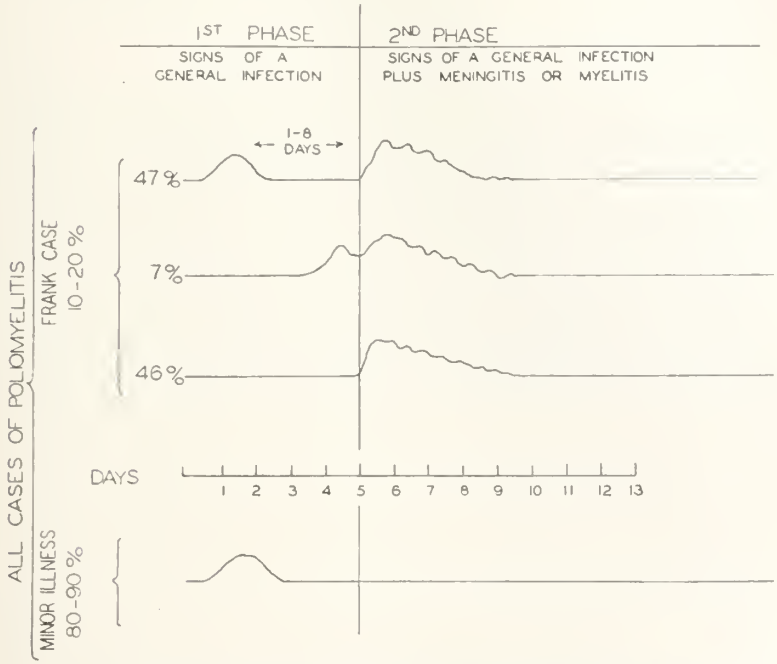

FIG. 1.—Schematic diagram representing distributional percentages which the various clinical forms of poliomyelitis may assume.

Within small communities the inclusion of the abortive case in one's survey may prove to be of great value. Most previous community studies on the spread of poliomyelitis have been dependent upon an adequate number of frank cases for statistical analysis. Over and above the fact that there must inevitably be large gaps in tracing the association between individual frank cases, such studies must also cover large groups of individuals, and as a result are generally beyond the scope of first-hand surveillance. On the

other hand, if the clinical boundaries of the disease are widened by the proper inclusion of abortive cases, one finds that a small epidemic of not more than 2 or 3 frank cases in a community may also furnish 10 or 20 abortive examples of the disease which represent adequate material for analysis and at the same time, if the community is small, first-hand contact can be made with the entire susceptible population in the community. In this respect another line of approach is open which may be useful for elucidating some of the obscurities which concern the spread of poliomyelitis.

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GENERAL REMARKS ON THE OUTBREAK OF POLIOMYELITIS IN PHILADELPHIA*

By J. NORMAN HENRY, M.D.
DIRECTOR, DEPARTMENT OF PUBLIC HEALTH, PHILADELPHIA

(Read by Courtland Y. White, M.D., Chief, Division of Laboratories, of the Bureau of Health.)

I HAVE been asked by Dr. J. Norman Henry, the Director of the Department of Public Health of Philadelphia, to present this paper at this time because of his uncertainty of attending this meeting. This task is difficult, as the administrative part of Public Health work is far from my training in medicine. At this time, however, I believe the work of the Department of Public Health should be thoroughly understood by the medical profession and especially in relation to the present situation of acute anterior poliomyelitis. Before going into details of the various phases of this work, may I present a word or so in reference to acute anterior poliomyelitis from the standpoint of a larger city.

Acute anterior poliomyelitis is a disease that has been among civilized communities for many years. Reference would indicate that possibly infantile paralysis existed in early Egypt, so examinations of the Egyptian mummies seem to suggest. To go back over the history of this disease is not the purpose of this paper; however, a few words in reference to the two major epidemics of this disease in which we are especially interested will not be amiss.

In 1916 the cities of New York and Philadelphia passed through epidemics of anterior poliomyelitis—the former with 8928 cases and Philadelphia with 1006 cases.

The epidemic started in New York City, and especially in the borough of Brooklyn, and a few weeks later had traveled toward Philadelphia and the Eastern States.

In 1931 New York City was visited by another epidemic of 4138 cases. Since New York City is only a few miles to the northeast of us, we had every reason to believe that Philadelphia would share in that epidemic.

* Read October 5, 1932.

In 1931 Philadelphia had recorded only 106 cases of acute anterior poliomyelitis, and when the cases from outside of Philadelphia are subtracted from this total, only a very slight increase of cases is to be credited here to this epidemic.

In this year of 1932 Philadelphia had recorded only 5 cases of acute anterior poliomyelitis to July 1, 1932, thus:

March 17, 1 case.
March 30, 1 case.
April 8, 1 case.
May 16, 1 case.
June 27, 1 case.

After July 1, 1932, the number of cases very soon began to increase, and by July 25 had reached the total of 15 cases so far for July, 6 of which were sent in from the outside of Philadelphia. It became evident that acute anterior poliomyelitis was headed for some sort of an outbreak.

On July 25, 1932, the Department of Public Health took definite steps to organize its resources for what emergency might take place.

On this date the Director, Dr. J. Norman Henry, with the assistant director, Dr. George A. Knowles, together with the chiefs of the various divisions of the Bureau of Health and the superintendents of the hospitals met in conference to outline the course to follow during the emergency.

The following outline of this action may be briefly stated:

A. Department of the Director

1. *Weekly Meeting* of the chiefs of divisions of Bureau of Health, and superintendents of hospitals, for the discussion of the situation and to make such recommendations as the emergency demanded.

2. *Monthly Bulletins* of the Department covering articles appropriate to an epidemic of acute anterior poliomyelitis:

1. Plans of the Health Department for the control of acute anterior poliomyelitis. Seth A. Brumm, M.D.,
Chief, Division of Communicable Diseases.
2. Early diagnosis of acute anterior poliomyelitis.
Louis Segal, M.D.
3. The use of convalescent serum and contact serum in the treatment of acute anterior poliomyelitis.
H. G. Fretz, M.D.

4. The methods for the collection of cerebrospinal fluid and the interpretations of the findings in acute anterior poliomyelitis. C. Y. WHITE, M.D.
5. The use of normal blood as a preventive measure in acute anterior poliomyelitis. DIRECTOR'S OFFICE.
6. The resolution of the Board of Health deferring the opening of schools and prohibiting the assemblages of children under eighteen years.
7. The management of acute anterior poliomyelitis at the Philadelphia Hospital for Contagious Diseases. P. LUCCHESI, M.D.
8. Acute anterior poliomyelitis from the viewpoint of a neurologist. GEORGE WILSON, M.D.
9. The technique of lumbar puncture. JOHN O. BOWER, M.D.
10. Map showing the distribution of the cases of acute anterior poliomyelitis in Philadelphia to date.

3. *Board of Health.*

1. The members of the Board of Health were notified frequently of the general conditions of the outbreak and their advice was sought.
2. On August 31, 1932, the Board of Health passed a resolution that prohibited the assemblages of children in public and semipublic places, and that all public, parochial and private schools remain closed until September 20.
3. On September 13, 23 and 30 and October 4, the Board of Health modified their previous rulings as the conditions warranted.

4. *Publicity.*

A. Newspaper—Daily news announcements giving number of cases reported and deaths. General advice to the public. Advice to camps. Request for donors of blood.

B. The Roster of Philadelphia County Medical Society was used for special announcements to the members of the medical profession and such statements as deemed of immediate value for the care of the cases.

C. The radio—Talks on infantile paralysis. Daily reports—How best to avoid assemblages, etc.

5. *Medical Staffs.*

A. Meeting of various hospital staffs. In which the chief resident physicians, visiting chiefs and pathologists met in July at City Hall Annex and the situation of a possible outbreak of acute anterior poliomyelitis was presented. A general discussion was presented on the subject of what the various hospitals could do—in case of an epidemic of acute anterior poliomyelitis, and the coöperation of the hospitals was sought and heartily given.

B. Diagnosticians, chiefly a group of pediatricians, were called. This group outlined the pediatric side of the subject and volunteered coöperation to help the Department of Public Health.

C. Orthopedic surgeons and neurologists. A large committee met and a subcommittee of this group formulated plans for the after-care of cases.

D. Dr. Simon Flexner, of New York, was frequently consulted and gave valuable advice on the subject. Dr. William E. Park was likewise consulted and gave valuable advice.

E. Emergency Aid was asked to give its aid and resources to see that no child would fail to receive the proper hospitalization and after-treatment if necessary.

B. Division of Communicable Diseases

A. 1. Plans for an adequate telephone service—receiving reports of cases—giving advice or answering queries. Keeping in touch with the field work of inspectors.

2. The collection and tabulation of data from cases reported.

3. Spot maps and graphs of the cases reported. Comparison of the maps and graphs of the 1916 epidemic.

4. Interviewing those interested in the disease from home and outside of Philadelphia.

5. Distribution of serum.

6. Enforcement of quarantine.

B. The organization of a corps of medical inspectors for the collection of blood from convalescent patients of previous years and immediate contact donors of former cases. This group bled 540 convalescent cases, 592 contact cases and 456 normal blood donors.

C. Division of Child Hygiene

1. Nursing corps solicited and interviewed and made arrangements for bleeding of donors of blood.

2. In charge of bleeding stations established at City Hall Annex. The nurses and parochial school inspectors taking charge of this work.

3. After-treatment of cases.

D. Division of Housing and Sanitation

1. The inspectors of this Division were used to check up on enforcement of the Board of Health resolutions governing the closing of schools and the assemblages of children under eighteen years of age.

2. Complaints handled by this division.

E. Division of Laboratories

A. Examination of spinal fluids.

B. Laboratory work for the Philadelphia Hospital for Contagious Diseases on all anterior poliomyelitis cases.

C. Preparation of serums.

1. Convalescent serum.
2. Direct contact serum.
3. Normal blood serum.

D. Telephone service for giving directions for the use of serums and whole blood.

F. Hospitals

A. Philadelphia General Hospital was used in the early part of the outbreak for the hospitalization of cases and admitted 49 cases. It soon became evident that the Philadelphia General Hospital would not have sufficient room to hospitalize all cases. The cases were then transferred to the Philadelphia Hospital for Contagious Diseases.

B. The Philadelphia Hospital for Contagious Diseases, on August 12, 1932, opened wards for the use of these cases and has admitted 322 cases.

Various other hospitals of Philadelphia have admitted these cases.

The outbreak of acute anterior poliomyelitis in Philadelphia this year has brought forth certain phases of this disease which may be especially commented upon:

1. Philadelphia, this year, is the only large city in the United States where any great number of cases of acute anterior polio-

myelitis has occurred. This has followed one year later than the larger epidemic in New York City in 1931. There has not been any known reason why a year delay has taken place.

2. The prompt and abrupt increase of cases in the month of July likewise cannot be explained. The distribution of the first 100 cases in the city is difficult to explain. There seems to be no seasonal temperature or any theory of contact transmission, either direct or indirect, which would explain this distribution. Direct contagion plays a part only in a very small number of cases, as far as can be definitely proven.

3. There have been to date 728 cases and 84 deaths. A case death-rate of 11.5 per cent. Compared to 1916 epidemic, where the number of cases was 1006 and the death-rate 30.5 per cent.

The number of cases reported showing paralysis or palsies or weakness of limbs is only approximately 54.7 per cent of the cases. In 1916 the great majority of cases showed some paralysis.

Conclusions from these figures cannot be drawn at this time.

It is generally believed that diagnosis of this condition is made much earlier this year than in 1916. Our reported cases undoubtedly include many cases of the abortive type.

4. It has been noted that the colored race has been more generally infected this year than in 1916.

In 1916 2 per cent of the reported cases were of the colored race.

In 1932 16.8 per cent were of the colored race.

5. The peak of the present outbreak seems to have been on August 24, 28 and 30, when 20 cases on each of these days were reported. Although on September 16, 19 cases were again reported. From this information the peak of the outbreak seems to fall in the late days of August or in the early days of September.

6. The age incidence is this year:

Under 5 years	415 cases
Under 10 years	181 cases
Under 15 years	86 cases
Over 15	47 cases

A suggestion from these figures that there are more in the upper bracket of figures than in 1916.

When one compares the number of cases infected with the population of the same age it will be seen that the percentage of the infected is about 0.1 per cent of this population. The rate of infection, therefore, is much less than in diphtheria, scarlet fever

and measles. Our apprehension in this particular disease is not so much from the number infected but from the appalling condition that remains after the outbreak is over.

As far as can be gathered at this time, about 60 per cent of the cases reported will either have died or show some form of infantile paralysis. That means that in this community about 400 children will be more or less crippled. Such a picture is very disheartening.

7. During this present outbreak we have advised the use of human convalescent and direct contact serum under the following conditions:

(*a*) After the diagnosis has been made or greatly suspected and before paralysis.

40 cc. of convalescent serum intravenously.

60 cc. of convalescent serum intramuscularly.

(*b*) If paralysis has been present and has existed for any time the use of serum has not been recommended.

(*c*) The serum has been recommended for use where paralysis has taken place and because of the fear of the further extension of the paralysis.

(*d*) The use of whole blood, obtained and injected immediately, from the parents of children was recommended in the hope of preventing infection. How much value this whole blood has been must be determined at some later date. It has seemed to be good practice to employ it. The blood from both the father and mother should be insisted upon for injection.

Under 6 months	6	.8 per cent
6 months to 1 year	37	5.5 "
1 to 5 years	372	51.5 "
5 to 10 "	181	24.5 "
10 to 15 "	86	11.5 "
15 to 20 "	33	4.5 "
20 to 30 "	10	1.3 "
30 to 40 "	1	.02 "
40 to 50 "	1	.02 "
Over 50 years	1	.02 "
	728 cases	

Under 5 years—approximately	57.0 per cent
Under 10 years—approximately	81.9 "
Under 15 years—approximately	93.7 "

Hospitalization December 31, 1932, 664 cases, 91 per cent
64 cases, home
664 cases, hospitalized
322 cases, Philadelphia Hospital for Contagious Diseases
49 cases, Philadelphia General Hospital
293 cases, other hospitals

The true value of human serums at this time cannot be determined in our present knowledge of the disease. It is probably true that the use of such sera, after the damage to the cord has been sufficient to produce a paralysis, are of no value. The value of such sera play in preventing infections, or modifying the infection after it has taken place, cannot be determined at this time. It is only after a great number of cases have been studied that our knowledge of the disease is better understood and the true value will be ascertained.

At present I (personally) would recommend the use of serum under the conditions above stated.

The antibody content of any blood is always questionable unless some means are at hand to readily test it. No practical method for this determination is possible at this time. It would seem good practice, therefore, to use only pooled sera from both convalescents and contacts for such treatments.

In conclusion, I believe if Dr. J. Norman Henry were here he would express his thanks to the various agencies, groups and individual physicians and various medical societies of Philadelphia for their unselfish coöperation during the period of this outbreak, and to the great number of people who have donated and repeatedly donated their blood for use in combating this outbreak.

A PRELIMINARY REPORT CONCERNING THE PROPHYLACTIC USE OF PARENT'S BLOOD DURING THE ANTERIOR POLIOMYELITIS EPIDEMIC OF 1932*

By HOWARD C. CARPENTER, M.D.

AND

JOSEPH STOKES, JR., M.D.

The authors have realized that whereas there have been many studies concerning the use of parent's blood or convalescent serum during the preparalytic stage of anterior poliomyelitis in human beings, very few investigations have been made concerning the use of blood or serum for the purpose of passively immunizing infants and children against the disease.

Dr. William H. Park[1] used whole blood from parents and convalescent serum in the attempted passive immunization of 200 cases last year in New York. The dosage used was 25 to 30 cc. of serum or 50 to 60 cc. of whole blood. Two of his contact cases developed the disease five days after injection and 1 six days after injection.

A large series of cases, however, to test such attempted immunization has been lacking. At the beginning of the present epidemic we felt that more information concerning the value of such injections might be obtained if a larger group of cases could be studied in a specified census area of the city. Our work was based upon the purely experimental hypothesis that immunization of infants and children against anterior poliomyelitis might be obtained in a manner similar to the method of immunization now commonly used against measles. For this reason it was decided to use 60 cc. of whole blood from one of the parents in the form of an intramuscular injection. We would have preferred the mixed whole blood of both parents, but it was obviously impossible to obtain both parents in most cases and we preferred a uniformity of technique.

The census tracts, outlined by the Bell Telephone Company

* Read October 5, 1932.

[1] Personal communication.

around The Children's Hospital, offered quite accurate statistics concerning this area of the city where the disease incidence was the highest. Although 1341 infants and children under ten years of age received injections at the Children's Hospital, only 620 children fell within the census' tracts chosen. These tracts had a population of 23,790 under ten years of age in 1930.

Among the 1341 infants and children 3 cases of poliomyelitis developed, 1 in seven days and 2 in ten days after the injection of the blood. All 3 of these cases had typical neck and back signs with a marked increase in the spinal fluid cell count. However, no weakness or paralysis developed in any one of them.

A fourth case was diagnosed by our own hospital as poliomyelitis on the basis of the signs present and a spinal fluid cell count performed at another hospital. This case was sent to the City Hospital for Contagious Diseases, where it was felt that the diagnosis was not correct and that it should not be called poliomyelitis. It is still our feeling that this case was a very mild or "abortive" poliomyelitis.

A fifth case, who had come in contact with a brother suffering from poliomyelitis, showed a febrile reaction six days after the injection of whole blood. The symptoms were more typical of secondary reactions to blood injections (which were not infrequent in this series) than of those of poliomyelitis. The spinal fluid cell count was 20 lymphocytes, and the case was transferred on the same day to the City Hospital for Contagious Diseases on account of lack of room at our own hospital. The physicians at the City Hospital did not feel it was a case of poliomyelitis and the signs and symptoms cleared up completely on the next day after admission. We feel that this case might have been poliomyelitis but in all probability was not.

In other words, there were 3 frank cases of poliomyelitis among the 1341 children injected, with no weakness or paralysis in any one of the 3.

We attempted to immunize as many children as possible in a certain district surrounding the Children's Hospital, so that we might compare the incidence in this group with the incidence in the district as a whole. We felt it was possible to use the children from one to ten years of age not injected as a control group. 620 children in this specified area were injected and 2 of these children developed the disease ten days after injection. The spinal fluid

cell count, signs and symptoms were typical, but no weakness nor paralysis developed. The incidence is, therefore, approximately 1 in 300 in this specified area, which was practically the same as the incidence of poliomyelitis in the entire census tract that we chose, according to the records of the Department of Health.

In the group of 1341 children injected, the incidence of 4 cases is about the same as the general city incidence. The group of 620 children was selected from the entire group of 1341 children according as to whether they lived inside or outside of the census tract chosen.

Injections were started on August 20. The epidemic began about the last week in July and reached its greatest intensity during the last week in August, the greatest number of cases being reported on August 28. Only 346 of the group received their injections from August 20 to September 1. The injections were practically completed, however, by the end of the first week in September. The epidemic remained severe throughout September and October.

The fact that none of the cases in our entire group injected suffered any weakness or paralysis could be considered as somewhat favorable evidence for the use of whole blood of parents. What evidence we have been able to obtain from the reports of other hospitals in the city have been also somewhat favorable, although we do not have a sufficiently accurate analysis of the children injected with convalescent serum or whole blood, outside of our own hospital group, to furnish a reliable report. We do not feel, however, that our work or the work of others has been at all conclusive since, despite the large number of cases injected, the disease incidence is so low that definite conclusions cannot be drawn.

THE FACIAL EXPRESSION OF THE EMOTIONS WITH SPECIAL REFERENCE TO VIOLENT EFFORT AND FATIGUE*

The Nathan Lewis Hatfield Lecture
Lecture XI

By R. TAIT McKENZIE, M.D., LL.D., R.C.A.
J. WILLIAM WHITE PROFESSOR OF PHYSICAL EDUCATION, UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA

Before taking up the subject of the evening I wish to give over to the safe-keeping of the College for their permanent collection four bronze masks cast by the lost wax process, the result of nearly thirty years of intermittent study, illustrating the facies of violent effort, breathlessness, fatigue and exhaustion. They are the third set that has been cast; the first set was used for exhibition purposes in connection with my other art work; and in a London gallery attracted the attention of the late Master of Christs College, Cambridge, the great biologist, Sir Arthur Shipley, who wrote in the Cornhill Magazine during the war an article on Hate, using the mask of violent effort as a frontispiece. He asked me to lend them to him when not on exhibition to hang on his study wall, a request I was glad to grant, and they were on his study wall when he died. When I wrote for them, the executors very properly asked me to prove ownership, thus giving rise to a very delicate situation, as no writing had passed between us on the subject; so I proposed to them that the four bronzes be presented to the Anatomical Department of Cambridge University by me in loving memory of my friend, which was readily agreed to by all parties concerned.

The second set belongs to the University of Pennsylvania, having been purchased by the J. William White Fund for research in physical education and athletics, and this is the third.

There are, however, examples of them in plaster at McGill University where the investigation was started, at the Royal College of

* Read November 2, 1932.

Surgeons, London, sent at the request of Sir Arthur Keith, D.C.L., D.Litt., D.Sc., Conservator of the Museum of the Royal College of Surgeons of England, and elsewhere.

As a practising athlete during my college days, and as a close follower of athletic competition, I was early intrigued by the facial contortions made by athletes during competition, as every observing spectator must be; and it was the desire to bring some order out of this apparent chaos that led to this study.

As a demonstrator of anatomy at McGill University the opportunities for making dissections of the facial muscles were abundant during the four hours a day that we spent in the dissecting room; and, seeing my interest in them, my chief, Prof. Francis J. Shepherd, suggested that I take his lecture hour on this subject before the class, a rare privilege eagerly grasped, and this became an annual event for the young demonstrator. This, of course, led to a search for what had been already done by others, and the discovery of the work of Lebrun, the court painter of Louis XIV, with his drawings of faces charged by emotion, which are not of great value; and to Lavater, in 1772, with his ponderous folios on Physiognomy; to Petrius Camper, in 1791, with his facial angle and lectures on expression, and also to Sir Charles Bell's monumental *Anatomy of Expression*, in 1806, filled with shrewd observation, illustrated by his own drawings and couched in his graceful and stately English.

The search discovered for me Duchenne, that French country doctor practising in Boulogne, who first used the electric battery to produce contraction of the facial muscles singly and in groups and so throw new light on this fascinating subject; and finally to Charles Darwin's *Expression of the Emotions in Men and Animals*, published in 1872, which summed up the subject as he alone could and gave that stimulus that only he could give to the young student wishing to make his contribution in this field of science.

It was in the company of these three men, Bell, Duchenne and Darwin, that the work was begun, but I must not forget Paul Richer, Professor of Anatomy at l'École des Beaux Arts, Paris, whose advice and experience were freely put at my service.

The facial muscles form a group by themselves, differing from the general muscular system in three particulars. They do not move bone on bone but the skin only, sometimes having no bony attachment at all. They have no sheath, but their fibers lie scattered in the subcutaneous fat, and they are frequently in action uncon-

sciously and not under the control of the will. They are specialized parts of the great panniculus carnosus sheet so active in the patient horse and cow in their vain attempt to shake off the persistent fly. It is displayed by the artistic butcher who cuts fancy patterns through its rosy sheet into the white of the subjacent fat to lure his carnivorous customers to further purchases. In man it is represented only by the platysma myodes and its differentiated fibers in the face.

The facial group is dominated by two purse-string-like, orbicular muscles, one that contracts and purses up the lips as in whistling, and the other, a double one about each eye, which closes the lids, lowers the eyebrows and produces the crows-feet at the outer angle of the eye. Practically all the others are antagonists of these two.

Over the forehead is the frontal producing the transverse wrinkles of surprise as the eyebrows are raised by it. Some individuals can contract the occipital belly of this muscle and move the whole scalp and also the rudimentary muscles of the ears, but this is only for the gifted few. The small corrugator supercilii draws the eyebrow up and inward obliquely like raising a curtain, making wrinkles at the base of the nose and center of the forehead, and registering pain. The root of the nose is creased by the pyramidalis in menace and rage.

All the other muscles of the mouth pull it up or down or out, against the action of the orbicular. One raises the upper lip and the nostril in grief. Another raises the upper lip at its outer end to expose the canine tooth in contempt or dislike, which is our poor attempt at what the dog does when he bares his fang as he walks stiff-legged and bristling toward his enemy.

The zygomatic muscle picks up the corner of the mouth and carries it out and up, curving the naso-labial fold into a smile. The man who is down in the mouth, who pulls a long face, is using the depressor of the mouth angle, and when he weeps he uses the depressor of the whole lower lip and a small muscle in the center attached to the jaw and to the skin of the chin, which is the first to tremble in the child about to weep, and which shoots out the lip of the disgusted adult.

The platysma itself is the muscle of emphasis. It turns the face of fear to terror, of pain to agony (Fig. 1), and of astonishment to horror as it pulls down the lip, opens the mouth and stands out in ridges from the chin to the upper chest. In the old, these "dewlaps" remain when the face is at rest and no makeup can conceal them.

Let us examine the action of these muscles under different emotional states to see just how they bear on the facies of violent effort and fatigue, taking up first the more pleasurable emotions that lead to laughter.

A smile begins by drawing outward and upward the angles of the mouth on both sides and a slight puckering of the eyes scarcely visible in youth. Much of the fine writing and controversy about Leonardo da Vinci's great masterpiece, the "Mona Lisa," hinges on the changing expression of her face, and much has been read

Fig. 1

AGONY

(From Duchenne's "Mechanisme de la Physionomie Humaine." This expression is artificially produced by electric stimulation of the muscles of expression.)

into it and written about it that would have astonished the artist himself. An examination of the picture shows that one side of the mouth is raised into a slight smile; the other is not; and if you concentrate the attention on one and then on the other the mood of this beautiful woman seems to change from moment to moment. To the layman this is a mystery just as is the fact that the eyes of a front face portrait have the disconcerting power of following you about the room.

The next stage of the smile is the creasing of the cheek and the puckering of the lower eyelid, so admirably caught by Rubens in his

quick sketch of laughter. (Fig. 2.) The expression is not complete without this action of the orbicular muscle of the eye. The two are closely connected, as you can see for yourself by winking hard and noting how the action is attended by an involuntary twitching of the lip.

When laughter becomes hearty, the respiration becomes involved. (Fig. 3.) The breath is taken in and expelled in gasps, the face becomes red, the blood-pressure rises, the eyes become engorged

Fig. 2

LAUGHTER

(A sketch by Rubens. A perfect interpretation of the associated action of the zygomaticus major and the lower part of the orbicularis of the eye.)

Fig. 3

MASK OF LAUGHTER BY JEAN CARRIES

(Illustrating the puckering of the eyelids and wrinkling of the nose which this state shares with rage and violent effort.)

and, to protect them from injury by rupture of the delicate blood-vessels, the orbicular contracts pressing on the contents of the orbit. Tears start into the eyes, and the laugher holds his sides, in agony —he nearly "dies of laughter"—and as the convulsion passes off, he wipes his eyes and slowly and with difficulty gets his breathing back to the normal as he calms down.

Laughter involves violent effort, and we see some points in common with it, especially about the eyes.

Let us now approach the problem from the path of pain which

first appears in the obliquity of the eyebrow drawn up and in by the corrugator. The outer angle of the lip (Fig. 4) is depressed, and as the emotion deepens the upper lip is raised near the center,

Fig. 4

SOLICITUDE

(Study by the author for the head of "Alma Mater" in the Girard College War Memorial. Note the slight obliquity of the eyebrows, the drooping of the corner of the mouth, the beginning of the expression of grief.)

the nostril is dilated and the stage is set for its most active manifestation, weeping.

Here again the respiration plays its part. (Fig. 5.) The weeping child gasps for breath, the air is expelled until the lungs seem to be empty. The eyes are tightly closed, the mouth open. Jacobs has

FIG. 5

THE WEEPING INFANT OF M. A. JACOBS

(Showing the frowning brow, the tightly shut eyes and the puckered eyelids that this expression shares with violent effort.)

admirably shown it in his head of the crying child with its swollen lids, down-drawn, open mouth and wrinkled nose. When the crying fit is over the redness of the face pales as the blood-pressure goes down and the breathing gradually returns to normal.

Fig. 6

MASK OF "VIOLENT EFFORT"
(By R. Tait McKenzie.)

Let us now examine the mask of violent effort in the light of what has been said. (Fig. 6.) This is the face of the sprinter or jumper or hammer-thrower. The breath has been caught and held until the end of the effort, for the 100 yards is run in one breath. In the face there is a general converging of the lines to the root of

the nose with transverse wrinkles over the bridge. The frowning brows are drawn down and the eye is narrowed to a mere slit as we found it in both laughing and crying where the compression of the heart and lungs by the muscular contraction of the chest walls drives up the blood-pressure to the point of seriously endangering

Fig. 7

VIOLENT EFFORT

(As seen in the broad-jumper whose eyes are tightly shut. Note the action of the levators of the upper lip and the platysma in strong action retracting both lips from the clenched teeth.)

the delicate bloodvessels of the eye from overdistention. The hammer-thrower or the jumper (Fig. 7) often does close his eyes at the moment of greatest effort, and the sprinter would if he did not have to keep his course.

The nose and upper lip have a snarling expression, the nostrils are distended and the lower lip drawn tightly across the clenched teeth except at the angles of the mouth where there are little pouches caused by the pulling of the platysma which stands out on the neck like cords.

Fig. 8

RAGE

(Original drawing by Sir Charles Bell in his "Anatomy of Expression" showing the wrinkled nose, frowning brow, and snarling nose and mouth, which it shares with violent effort. It differs from it in not having the eyes open and staring.)

The general expression of the face corresponds to the face of rage as sketched by Sir Charles Bell except that in his picture the eyes are more widely opened and the lips less retracted (Fig. 8), and in all these states there is a rapid rise of the blood-pressure which effort shares with both laughter and tears.

The athlete is possessed by a sort of physical rage, and the intensity of his concentration is mirrored on his face, which indicates the fury of his effort.

Many artists have depicted fury. (Fig. 9.) Rude, in his Marseillaise group on the Arc de Triomphe, shows it in the furious hel-

meted figure with upraised sword urging the soldiers young and old to water the furrows of their land with the blood of her enemies. Michelangelo shows it in a head with contracted brows, flashing

Fig. 9

FURY—"LA MARSEILLAISE" BY RUDE

(From the group in the "Arc de Triomphe," Paris. Showing the frowning brow and snarling nose of effort, but with the open, shouting mouth.)

eyes and open shouting mouth, but they have little in common with the mask of violent effort except in the brows.

From fury it is a short step to horror and fear, and when fear enters in we pass to anxiety, and anxiety may be physical or mental. Perhaps the most familiar form of this emotion may be seen in the

hospital ward as we look at the patient whose heart is laboring to keep up with its vital work or whose lungs are barely able to keep from suffocating the spark of life, in a double pneumonia. The "muscle of pain" now draws the brows into the obliquity of pain and sorrow. The eyes are half-closed, the corners of the mouth are drawn down and the upper lip slightly raised as the nostrils dilate for the intake of each breath. The patient is in dread of suffocation. Fear is in his heart and on his face. It is the same fear that is on the face of Laocoön, the Trojan priest who advised against the receiving of the wooden horse sent by the Greeks. Athena in revenge sent two serpents from the sea who wound themselves around him and his two sons as they were sacrificing on the shore, and the sculptor shows him struggling vainly to free himself from their coils; fear, anxiety and approaching death all show in his face, the oblique eyebrows, the gasping mouth and with it the upturned eye of exhaustion. In actual suffocation by hanging, the face is different, for here the circulation is suddenly stopped. Strangulation is well described in Henry VI, Part 2.

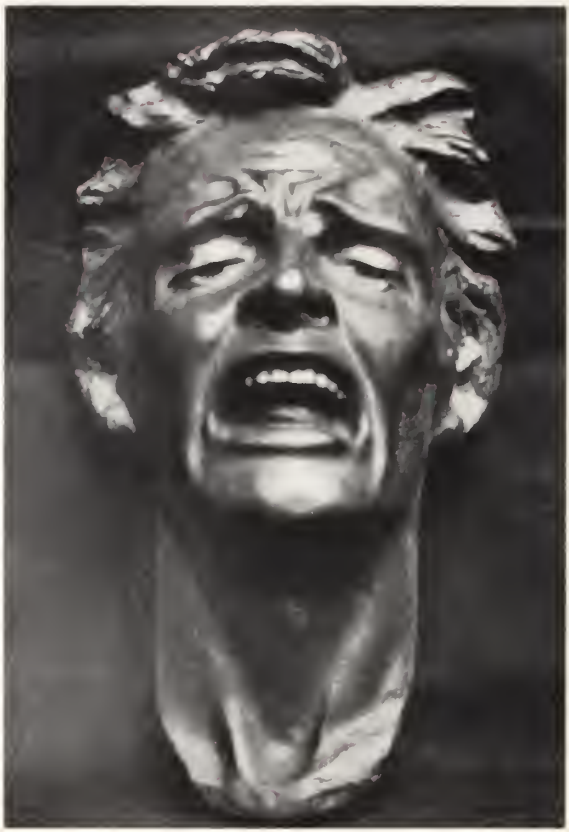
> "But see, his face is black and full of blood,
> His eyeballs further out than when he lived
> Staring full ghastly like a strangled man
> His hair upreared, his nostrils stretched with struggling."

How much of mental horror does such an expression indicate? Not much, I fear. It is due more to the damming of his circulation than the damnation of his soul. Mantegna shows it in his drawing of Hercules strangling Antaeus in his powerful arms. The athlete does not get to such an extremity. After the first exhilaration he feels an increasing uneasiness in his chest during a long race. This becomes more and more marked. He feels as if an iron band were clamped about it and as the struggle for breath becomes more and more acute his distress and anxiety increase until they are all but intolerable. The second mask of "Breathlessness" shows the characteristic facies. (Fig. 10.)

The general direction of the eyebrows is just the reverse of that seen in violent effort. They are drawn upward and inward by the corrugator, as seen in all mental distress, grief or bodily pain. The upper lids droop and half cover the eyeball, giving a look of great lassitude to the suffering expressed in this region. The nostrils are widely dilated and the mouth gapes with lips retracted in the mad struggle for air. The raised upper lip adds to the look

of sorrow and pain while the down-drawn mouth, the tongue closely pressed against the teeth and the sunken cheek all go to increase the exhausted, haggard look, characteristics of this state in distinction to mere bodily pain or mental suffering. The general poise of the

Fig. 10



MASK OF BREATHLESSNESS

(By R. Tait McKenzie.)

head is backward. (Fig. 11.) The chin is thrust forward and the neck is strained.

The Marathon Race is the culminating event of the revived Olympic Games, and at Los Angeles the stadium was crowded to see the finish of this hard 26-mile grind. The competitors were

Fig. 11

BREATHLESSNESS

(As seen in Meredith at the finish of the half-mile race. Note the raised, oblique eyebrows, the lowered eyelids, the open mouth, and the retracted lips.)

to enter the stadium through a tunnel and run about three-quarters of the way around the 400-meter track to the finish. The bugles sounded as the first man emerged upon the track and the slight form of Zabala, an Argentine, appeared. He was very tired, but the stimulation of the cheering crowd made him quicken his pace and he finished almost sprinting. (Fig. 12.) I have never seen such an expression of pain and breathlessness on a face before. The obliquity of the eyebrows, the drooping of the eyelids and the drawn lines of the mouth were vividly typical as he was walked about scarcely able to stand, supported by the secretary, Zack Farmer, and an attendant.

When the urgency of breathlessness passes, when the respiration catches up with the elimination of waste through the lungs, the athlete has a respite, partial, but quite definite from his pain and distress. In common speech he gets his "second wind" and he goes on for the third quarter of the mile fighting an increasing lassitude, the lassitude of general fatigue.

This is shown in the third mask, "Fatigue." (Fig. 13.) Group after group of his facial muscles begin to fail. A slight frown replaces the acute pain shown by the eyebrows. The eyelids seem heavy as with sleep. The upper lip is still slightly retracted and the mouth opens as the jaw drops. The cheeks are relaxed and flaccid and the whole face assumes a drunken and stupid expression. It is the face of intoxication. He could not pucker his lips in a whistle nor raise his heavy-lidded eyes; in fact, much of his failing attention is directed to keeping them open at all. It is in this drunken and stuporous condition that a man may swerve in a race and commit a foul quite unintentionally. He concentrates his mind with increasing difficulty until at last just before collapse we see the expression shown in the fourth mask, "Exhaustion." (Fig. 14.)

Here the frontal is in strong action. Up to this time it has not been used. Now it springs into action to draw up the closing and paralyzed eyelid. It gives a look of surprise to the forehead which the athlete is far from feeling. The lower part of the face remains unchanged, but to give additional assistance the head is thrown back so that he looks down his nose (Fig. 15) and is held balanced in this position, which requires the least muscular effort and so helps in this last effort to fight off collapse.

This balancing of the head is common to other conditions. Van Dyck shows it in his picture of a sleeping child. It is seen in the dying Alexander and in the head of Laocoön as the poison of the serpent's bite begins to take effect.

FIG. 12

BREATHLESSNESS AND FATIGUE

(As seen in Zabala, the Argentine, winner of the Marathon Race at the Olympic Games in Los Angeles, August, 1932. Photograph taken just after crossing the finish-line. Note the pain in the oblique eyebrows, the drooping of the eyelid, and the retracting lips of breathlessness and fatigue.)

Fig. 14

MASK OF "EXHAUSTION"
(By R. Tait McKenzie.)

Fig. 13

MASK OF "FATIGUE"
(By R. Tait McKenzie.)

In the athlete when this last flicker of consciousness is exhausted a marked change occurs. The dusky color of the skin becomes a

Fig. 15

EXHAUSTION AND FATIGUE AT THE END OF A MILE RACE

(The leader shows the loose, open mouth of exhaustion with the head thrown back and the eyebrows raised to see his course because the eyes are almost closed. The second runner shows the slight frown and drooping eyelids, and the loose cheek and mouth of fatigue.)

greenish-white. The circulation fails, the eye closes, the face becomes a blank, and he collapses on the track or into the arms of his friends. If we draw up his eyelid it will be seen that the eyeball

turned upward. The nostrils are pinched and the eye is deep in its socket. For the time being he is dead. It is the little death of syncope.

The expression of the face in natural death is always a blank, and the features settle into their habitual positions of relaxation. The smile in death is the habitual smile that was characteristic of the living. As the Persian sage puts it:

> On Parent's knee a naked new born child
> Weeping thou sat'st while all around thee smiled
> So live that sinking to thy last long sleep
> Calm thou mayst smile while all around thee weep.

Fortunately, the syncope of exhaustion is short, and the athlete quickly recovers. The heart resumes its function and a healthy color comes back to his face, and he is ready again to show in a future contest the rage and fury of violent effort, the pain and anxiety of breathlessness, the drunken stupor of fatigue and the equivocal pained pseudosurprise of exhaustion.

A SUMMARIZATION OF THE ADVANCE IN OUR KNOWLEDGE OF SOME FEATURES OF CANCER*

The Mütter Lecture

By H. GIDEON WELLS, M.D.

In considering the present state of our knowledge of cancer I have an undesirable advantage, or an advantageous disadvantage, in that my years of observation carry me back beyond the beginning of the present century and into the dark ages of cancer research. My induction into cancer research occurred in 1896, when I was appointed pathologist in the surgical clinic of Nicholas Senn, who shortly before had written the first comprehensive work in English on the *Pathology and Surgical Treatment of Tumors*. As a result many desperate cases were sent to his clinic from all over the country, and there I saw monstrous growths such as I have never seen since and which are nowhere seen nowadays except by medical missionaries and others who practise medicine in countries as medically benighted as was this country in 1896. So when I hear it said, and that not infrequently, that after all we really know very little about cancer, I feel much more cheerful about our progress than do those who do not realize how much we have learned in these past thirty-five years.

It is fair to say that cancer research had come almost to a point of complete despair at the end of the nineteenth century. Pathologic anatomy had established the relation of cancer tissue to normal tissue and had provided a basis for descriptive classification which permits of indefinite division into groups and subgroups, an entertaining process that is still going on, whether profitable or not. Bacteriology had exhausted its first enthusiastic expectation of settling the nature and cause of cancer, as it had triumphantly done with so many other diseases, and was beginning to think that after all there might perhaps be something in what the pathologists said about cancer probably not being a specific infectious disease. Pro-

* Read December 7, 1932.

tozoölogists had become a little weary of working out the life cycles of the protozoa that caused cancer, after disillusionments that shattered pride and dispelled vanity. Chemistry had found its feeble attempts to determine something chemically characteristic of cancer to be futile, and virtually abandoned the subject. There seemed to be no particular way for advance in the study of one of the most important of human diseases. What was being done on cancer research in the nineties was mostly in the nature of grasping at straws.

The renaissance of cancer research came when it was found that laboratory animals could be made to have cancer, more or less at man's will. This made it possible to put cancer research on the basis of an experimental science, with planned investigations in which the vitally important matter of adequate control could be provided for.

Three major steps have marked the early stages of the study of cancer by experimental methods. The first of these, and the one which put a fresh breath of life into despondent cancer research, was the demonstration, at the beginning of this century, by Jensen in Copenhagen and Leo Loeb in Chicago, that carcinoma and sarcoma could be transplanted and retransplanted in mice and rats for an apparently unlimited time. In fact, Jensen's mouse carcinoma is still being transplanted, over thirty years after its original removal from the mouse in which it started. The cells of this cancer now growing in laboratory mice are direct descendants of the cells of the original Jensen cancer mouse. As the maximum age reached by mice is about three years, this fact suggests that cancer cells are immortal, as apparently all cells are if they can be freed from the limitations of life of the organism as a whole. Carrel has carried connective-tissue cells eighteen years in tissue cultures in his laboratory.

The great expectations we had at first of knowledge coming from the study of transplanted cancer have not been realized, because, as was slowly appreciated, a transplanted tumor is fundamentally something entirely different from an autonomous primary tumor, in which the growth is derived from the cells of the animal in which it is growing. The transplanted tumor is always composed of foreign cells against which the host puts up more or less of a reaction, often successfully, so that with most transplanted tumors there is a marked tendency to spontaneous regression and healing, in con-

trast to autonomous cancers. Hence therapeutic results obtained with transplanted tumors cannot be safely applied to the problem of the treatment of spontaneous tumors in man, and heredity studies with transplanted tumors are probably of little or no significance in respect to the relation of heredity to spontaneous tumors, since it is quite a different matter whether an animal's tissues resist growth of transplanted cells or develop malignant cells themselves.

Although we now realize that the amount we can expect to learn about cancer from transplanted tumors is quite limited, because of these fundamental differences between transplanted and spontaneous tumors, nevertheless this discovery was of tremendous importance because it stimulated interest in experimental cancer research and yielded not a little valuable information about tumor cells. The chief result seems to be the demonstration that tumor cells are essentially the same things as the tissue cells from which they arose. Transplantation of tumors is no different from transplantation of normal tissue cells, and the same requirements are found to be necessary in each case. That is to say, transplantation is most likely to be successful when made into the same animal as the one which furnished the grafted tissue; grafts into members of the same family, *i. e.*, brothers, sisters or parents, have a much better chance of success than grafts into unrelated members of the same species, and grafts into other species are usually impossible. Such a striking dependence on close genetic relationship is entirely in disharmony with the hypothesis that cancer depends on a specific cancer parasite, for we know of no infectious disease transmissible from one animal to another that requires any such close relationship of the donor and the recipient of the infection.

The old suggestion that the cancer parasite is the cancer cell itself has received strong support from the study of transplantable tumors. Any procedure that destroys or devitalizes the cancer cell interferes with its transplantability, just as is the case with the transplantation of normal cells. No one has yet succeeded in separating from a *mammalian* carcinoma any agent that will produce a cancer in other animals, except viable cancer cells.

The so-called "Rous sarcoma" of chickens, which is transmitted by some agent passing through filters fine enough to hold back bacterial cells, presents unsolved problems that I have not the time to discuss here.

Transplanted tumors have furnished much material for the study

of immunity, specific serological diagnosis of cancer and treatment, with results that sum up to nothing encouraging when only the best controlled experiments are considered. Unfortunately many incautious and inexpert experimenters have aroused unwarranted excitement concerning supposed curative measures based on a small number of experiments, disregarding the common tendency for many sorts of transplanted tumors to disappear spontaneously.

More important, presumably, was the discovery that primary spontaneous cancer, altogether like cancer of man, can be made to arise in laboratory animals under the influence of chronic irritation of tissues or because of inherited properties. In 1914 Yamagiwa and Ichikawa in Tokio reported that squamous-cell carcinoma could be produced in rabbits by long-continued application of tar, a method and principle that have since been greatly extended in other laboratories and of much greater experimental value than the earlier observation of Fibiger that cancer may develop in the stomach of rats from the irritation produced by nematodes coming from cockroaches.

Early observations suggesting that heredity might play a rôle in the occurrence of spontaneous tumors in animals were followed by the more extensive observations of Loeb and Lathrop on subcutaneous (chiefly mammary gland) tumors, and of Maud Slye, who first showed on a large scale, with mice of known ancestry, how important a rôle heredity plays in producing tumors of various types and in different tissues. This work has established the fact that, by proper breeding, strains of mice and rats may be obtained with a high incidence of tumors, even independent of the application of any unusual stimulus or other factor to cause tumors to appear.

Since modern cancer research is now largely based on these three sets of observations, the question of animal cancer becomes a vital one in a consideration of the present and future developments of cancer pathology, etiology and treatment. A fundamental question is, What is the relation of the disease or diseases called cancer in animals to cancer in man? Is it the same thing or merely a similar thing or even something essentially different? Another question that may well be raised is, Can anything be learned about the nature of cancer in man by the study of neoplastic diseases as they naturally occur in animals?

The extensive study recently given to these growths has served to remove all doubts that they represent fundamentally the same

disease, cancer, in animals and man, and that the differences observed depend either on anatomical peculiarities of different species or on other differences between species, for the frequency of each type of cancer varies with each species. The identity of animal neoplasms with those of man is shown by the following facts.

The cells of malignant animal tumors resemble fundamentally those of human cancers, in that they are of embryonal character, showing many mitoses, which are often atypical, forming at times atypical cells differing in appearance from those of normal tissues, arranged atypically, infiltrating the adjacent tissues and destroying them, invading blood and lymph vessels to produce tumor emboli and metastases in remote organs. No infectious agent can be found in them, and they are no more contagious than human neoplasms. Animal carcinomas also exhibit the same characteristic age distribution as human carcinomas, rarely being seen before the middle age period and increasing in frequency with advancing age. Those tumors which exhibit histological malignant characters are as inevitably fatal as human cancers, recurring after removal and undergoing necrosis with ulceration and secondary infection like carcinoma in man. Especially in Miss Slye's large stock, with vast numbers of animals permitted to live out their natural lives, it has been found that even in mice, cancer is by no means limited to the readily observed carcinoma of the mammary gland. In fact I have seen in Miss Slye's mice most of the forms of tumors that I have seen in men, even rare tumors, such as a papilloma arising in the ependyma of the lateral ventricle, a malignant adenomatous growth of the anterior lobe of the hypophysis infiltrating the meninges and brain, and primary liver-cell carcinomas. Furthermore, each type of growth behaves about the same as it would in man. For example, the solid tumors of the ovary are commonly bilateral, the squamous-cell carcinomas of the mouth arise at points of irritation from broken teeth, etc., and I have seen them infiltrate the skull and invade the meninges. Basal-cell carcinomas, most often seen on the faces of old men, in mice usually are found on the skin of the face of exceptionally old individuals, and, like human basal-cell carcinomas, they do not readily produce metastases. Furthermore, the types of tumors which are histologically like benign tumors in man behave as benign tumors. Uterine fibroids are found in mice as benign growths, prone to calcify; even infiltrating adenomyomatosis is seen in the uterus of mice. Teratomas are seen chiefly in the ovaries, both in

mice and women, and typical hypernephromas are found in their kidneys. Thyroid tumors, alike in mice, dogs and men, have a strange tendency to look as if made up of a mixture of carcinoma and sarcoma, a feature not often seen in tumors from any other tissue.

Cancer is certainly not an exclusively human affliction, as has been especially well demonstrated by the comprehensive studies from your Philadelphia Zoölogical Society by Dr. Herbert Fox, who has published valuable reports of tumors of varied nature occurring in a great number of different species of animals. Indeed, the more we study comparative pathology the better we realize that there is nothing exceptional about man in respect to cancer, not even its frequency. Our idea that cancer is especially common in man is correct, simply because man is one of the few species of animals that is commonly permitted to reach old age, and cancer is a disease of old age. The few species of animals that often reach old age, as turtles, elephants and the rhinoceros, seldom have the benefit of clinical study or postmortem examination to tell us just how often cancer has been present. Systematic study of old dogs shows that neoplasms are extremely common among them; in fact, it is rare to find a really old dog without some sort of tumor, either benign or malignant or both. Multiple tumors are especially common in old dogs. It seems safe to say that tumor formation in dogs is as common as, and probably more common than, in men of corresponding age. Indeed, it seems probable that if one were to study a large group of mice, cows, dogs or horses, it would be found that they exhibited much the same frequency of neoplasm formation as a group of human beings in the corresponding part of the life cycle.

Cancer is undoubtedly a universal disease in multicellular organisms. It has been found in almost all species of animals that come under the observation of people able to recognize and report it.

As yet, as far as I can learn, the presence of true neoplasms has not been demonstrated in any invertebrates, except possibly the insects, but it is to be expected that they will be found as more examples of lower animal forms come before persons competent to recognize neoplasms in them.[1] Metcalf has even suggested that irregular and atypical nuclear division observed in protozoa may

[1] The early reports of a tumor of the pericardium of an oyster, observed by your great Leidy and reported by Ryder, and of an "adenomyoma" arising in a fresh water mussel, are not adequately described to permit us to know whether they were true neoplasms or not.

be equivalent to tumors in multicellular forms. The growing number of observations on cancer in animals adds strength to the statement of Bashford, in 1904, that "the great diversity of food, habit and conditions of life generally in animals in which cancer occurs shows that such external agencies have no causative influence." In Miss Slye's mice, kept under identical conditions of nourishment, temperature and environment, the incidence of cancer in different strains varies from zero to practically 100 per cent in mice of sufficiently advanced age. Certainly comparative pathology offers no support to the hypothesis, often advanced, that cancer depends on diet, and that it may be controlled by diet, especially vegetarianism. Most of Miss Slye's mice are on a vegetarian diet, and the vegetarian carp seem to offer as many tumors as carnivorous fish. Old cows and horses frequently develop malignant neoplasms, these being the chief examples of herbivorous animals permitted to reach old age under conditions in which tumors are likely to be detected.

Between different species there is, however, a marked difference in the frequency of different types of neoplasm, which presumably is of significance, but for which we still lack any explanation. For example, in man cancer of the stomach undoubtedly takes first place in frequency, but in every other species, as far as is now known, is extremely rare. Why this should be is an interesting problem. Dogs, with their high incidence of cancer in general, eating much the same food as their masters, do not have gastric cancer. There are but one or two reasonably authentic reports of cancer of the stomach in dogs. Possibly this depends on the fact that man is the only animal who eats hot and spiced foods. Many things can be cited in favor of this theory, but an attempt made in my laboratory, by Julian Lewis, to produce cancer of the stomach in dogs by feeding hot food was unsuccessful, though this negative result, of course, proves nothing.

Cancer of the cervix uteri is also relatively infrequent in other animals than man, although cancer of the corpus uteri is not so infrequent as cancer of the stomach; especially is it found in cows and rabbits, but even with these species it is a rare condition. Uterine carcinoma is seen more often in multiparous than in nulliparous women, but most other mammals are much more multiparous than women.

These facts suggest that the differences in occurrence of different neoplasms in different species depend not so much on the amount or

character of traumatism to which their tissues are subjected as on the properties of the tissues themselves, as determined by heredity. Supporting this view is the species distribution of cancer of the breast. Among mammals in general the mammary gland is one of the common sites of cancer, possibly because it is a highly specialized organ developed relatively recently in animal evolution. But of all the mammals the one with the most traumatized, overworked and specialized mammary gland, the dairy cow, seems freest from mammary cancer. I cannot find a single record of a cancer in a domestic cow's mammary gland, despite the fact that milk cows are commonly kept until they have reached sufficiently advanced years to be subject to carcinoma. None has ever been seen in the vast material coming to the Union Stock Yards in Chicago, according to the statement of the government pathologist, Dr. L. E. Day. Carcinoma is not particularly uncommon in the less-used mammary gland of mares.

Only on the basis of hereditary species susceptibility can be explained the facts that in mice the malignant tumors, especially of the mammary gland, are predominatingly carcinomas, whereas in rats the tumors are mostly sarcomas, and that in dogs the tumors especially seen are mixed epithelial and cartilaginous growths in the breast, thyroid tumors (often of mixed structure) and lymphomatous growths. So, too, we note that swine are especially prone to exhibit embryonal adenosarcomatous tumors of the kidneys, that horses show a high incidence of testicle tumors, considering how few old horses there are with testicles, that fowls are especially likely to have ovarian tumors, that gray horses are particularly subject to melanotic tumors of the skin and that sheep and cows have tumors of the liver most frequently. Another striking fact is that gliomas of the brain, so common in man, are unknown in other species, only one reasonably satisfactory report of a cerebral glioma, this in a dog, having been found in the literature.

These pronounced variations in susceptibility to cancer of different tissues in different species seem most readily explained as a difference in the inherent properties of the tissues, which suggests their dependence on inherited qualities that determine species differences. For example, mice very frequently suffer damage to the liver from parasites and from infections, but I have never seen or heard of a primary sarcoma of the liver in mice. Yet rats of certain families

very frequently develop sarcoma in the liver at the site of parasitic invasion. Other families of rats do not exhibit sarcoma of the liver when infested with parasites, thus resembling the mice. Furthermore, certain strains of mice develop epithelial tumors of the liver independent of parasitic invasion, whereas most other strains of mice never develop liver tumors under any circumstances. That is to say, the facts brought out by consideration of the comparative pathology support the view that the inherited constitutional makeup of the species is an important factor in determining what sort of tumor will occur in that species. A human analogy is furnished by the markedly greater tendency of Negroes to develop fibroid tumors and keloids than whites.

What seems to be the most useful step yet made in the study of cancer is the demonstration that laboratory animals can at will be made to develop cancer of their own tissues, just as human cancer is produced. We now have two methods that permit us to secure large numbers of animals with true autogenous neoplasms. One is by selective breeding, depending upon the demonstrated influence of heredity upon the occurrence of cancer, a matter that will be discussed more fully later. It is gratifying to find that research workers are now beginning to test proposed therapeutic procedures on natural spontaneous cancers secured by selective breeding, as reported in the paper by Itami and McDonald on the treatment, with negative results of mouse cancer by means of an active extract of adrenal cortex.

The other method of getting cancer animals, more popular because easier and less time-consuming, is by using the recognized factor of long-continued irritation in the production of cancer. This irritation may be produced by animal parasites, as in the production by Fibiger of cancer of the stomach in rats infested with a nematode; or a sarcoma of the liver in rats infested with a cestode, as observed in the Crocker Laboratory, New York. These methods are of limited applicability because they introduce an unusual, special and not readily controlled factor, a specific animal parasite. By far the best method yet devised was described, in 1914, by Yamagiwa and Ichikawa, who succeeded, where others had failed, in producing cancer of the skin of rabbits by the repeated application of tar, analogous to the tar cancers observed on the skin of man.

So readily are skin tumors produced in mice with tar that the

Tworts have been able to report on 4000 such growths. Although this line of study is only in its infancy, and much is expected of it with time, certain results seem already to have come from it.

One important result is added support for the now widely accepted view that cancer does not depend on a specific parasite. Advocates of the theory of a cancer parasite have always argued that the reason trauma and chronic irritation predispose to cancer is that they open the way for the supposed infectious agent to enter the tissues. But Yamagiwa produced carcinoma on the ears of rabbits by the application of a strongly antiseptic tar, and no one had ever before seen a carcinoma arise on the ear of a rabbit, despite the vast numbers of these animals under observation in laboratories, nearly all with plenty of openings in the skin of their ears to permit the entrance of cancer germs, due to needle punctures, ear tags, identification notches and rough handling.

The fact that an antiseptic agent, characterized by its stimulating cells through mild irritation to repeated multiplication, produces cancer, is in full harmony with the long widespread impression that cancer does not depend on any specific cancer parasite, but that it may be the result of any condition that repeatedly stimulates cell proliferation until the proliferative activity dominates the life processes of the cell, which acquires an exaggerated "habit of growth." The fact that the growth always appears at points irritated by the tar, whether this be the skin, the lungs or the stomach, also confirms the long-prevailing view that cancer is essentially a local disease and not a constitutional disease with local manifestations.

The importance of heredity in the genesis of cancer is also confirmed by the tar experiments, for there is a vast difference in the susceptibility of different species of animals to the production of tar cancer. While the skin of rabbits, and especially of mice, will often develop cancer after sufficient tar painting, it seems to be practically impossible to produce skin carcinoma in rats, guinea-pigs, dogs or fowls with tar. Nevertheless, carcinoma has been produced by tar in the uterus, urinary bladder and stomach in rats, and in the gall-bladder in guinea-pigs. Furthermore, sarcomas have been produced in rats and fowls by subcutaneous tar injection. These facts indicate an inherited species-characteristic difference in the susceptibility of different tissues to malignant transformation by a common agent. The fact that some animals of a susceptible species develop

tar cancers quickly, some slowly and some not at all, indicates that there are also individual differences in susceptibility. This is, of course, quite the same as the differences in susceptibility of *x*-ray-workers or dye-workers to cancer produced by the agents involved in their occupations.

The observation that the same agent may produce either sarcoma or carcinoma also affords evidence that these diseases are essentially the same. The apparent shading off of sarcomatous proliferation into inflammatory proliferation is so confusing that we have felt far from certain that we might not some day learn that all sarcomas were inflammatory processes and essentially different from carcinomas. The experimental results point in the other direction.

The experimental production of cancer by tar has thrown much light on the relation of age to cancer. It has been found that young animals are fully as susceptible to the action of tar as older animals, and perhaps more so, but at any age considerable time is required to produce a cancer with tar. Some animals will develop carcinoma in a few weeks, but some require a year or even more. Obviously, since young mice can develop cancer as well as old mice, senility of the tissues is not the reason why cancer affects mice and man chiefly in middle or old age, as we have commonly assumed. The explanation, as shown by the experimental work, is that a prolonged period of chronic irritation is usually necessary to produce cancer. It requires about one-sixth of the normal span of a mouse's life to produce tar cancer, which would correspond to ten to fifteen years of a human life. This fits perfectly with the experience with occupational cancer from dyes, *x*-rays, tar and other cancerogenic agents, for the cancer rarely appears until seven or more years have elapsed after the occupation has been begun; more often fifteen years are required, and it makes little difference at what age the victim began his disastrous occupation. These facts suggest that cancer of the stomach, breast or uterus, arising in a woman at the age of forty-five years, owes its origin to a slow, continued process which began ten, fifteen or even more years before, and not usually to some event immediately preceding the growth. Cancer is, then, not essentially a disease of old age, dependent on senescence of tissues, but a disease of long-irritated tissues, the necessary duration of irritation being seldom reached in the first half of life. This also seems to explain why authenticated cases of cancer following a single trauma are so rare.

The period of time that must elapse before cancer develops in mice subjected to tar painting varies greatly; it varies from three months to a year or more, corresponding to variations in man of from seven years to thirty years. This indicates that among different individuals of the same species considerable differences in the susceptibility to cancer exist, and has its counterpart in the clinical experience that occupational cancer appears only in a fraction of the individuals following a given occupation. It is to be noted in this connection that the types of malignant neoplasms that seem to depend on protracted stimulation by mild injury, as skin cancer and gastric cancer, rarely appear in the first half of life. The malignant tumors that are seen in childhood, such as the retinal neuroblastomas, cerebellar gliomas, mixed renal tumors and suprarenal neurocytomas are largely those that seem to arise independent of chronic injury, perhaps owing their occurrence to developmental defects or hereditary factors. The trend of these results of recent cancer research is in support of the old view that cancer is usually the result of protracted stimulation of tissue growth by nonspecific agents acting on tissues the susceptibility of which is determined by their hereditary background.

In seeking to learn whether the occurrence of cancer in man is in any way dependent upon his heredity, the usual method, until recent years, has been to compile statistics on the number of relations of cancer patients who exhibit cancer as compared with the relations of control subjects who did not have cancer at the time they furnished the information for the statistical study. Many such studies have been published, clinical histories of the patients of large hospitals generally having furnished the material used for analysis. Critical consideration of such contributions at once shows that they must be worthless because of the numerous sources of error in the material furnished for statistical study. The usual casual clinical histories are extremely unreliable, not checked by adequate investigation or cross-examination. Even if the facts were more carefully gathered the results would probably be quite as unreliable, because the average cancer patient before coming to hospital has discussed the matter with members of the family and is quick to answer questions as to cancer in the family, whereas the control subjects, who have entered the hospital for some other condition, have much less information to impart. It is not strange, in view of these facts, that almost invariably such statistical studies

have indicated that cancer patients have more relatives with cancer than patients who do not have cancer, and, therefore, that the impression long prevailed that heredity did play a part in determining the occurrence of cancer, although apparently not a very important part, since many persons with cancer knew of no relatives with cancer, and many persons with cancer had children who did not develop this disease. Still another error lies in the fact that the controls in most of these studies consist merely of persons selected as not having cancer at the time the history is taken, without regard to the fact that presumably 10 per cent or more of them will develop cancer at some later date. Family-history records can have no value in the study of Mendelian inheritance if they contain even a single error in an entire family tree, since this would lead to entirely erroneous conclusions as to the method of inheritance. Hence existing human-cancer statistics and records with their vast inherent error cannot possibly serve to solve the problem of whether and how heredity influences the occurrence of cancer.

But if statistical studies cannot furnish useful information, other sorts of evidence are offered by human material that are at least suggestive. One of these is the recognized occurrence of cancer families. Some of these families have become classical in cancer literature; such is the Bonaparte family, for Napoleon I, his father, his brother Lucien and two of his sisters, Pauline and Caroline, all were believed to have died of cancer of the stomach. One of the most remarkable is the family of Madame Z., reported by Broca in 1866, and mentioned in virtually every discussion of heredity in cancer since that time. Many more such families might be described. The chief trouble with most of these reports is that they do not include all the noncancerous members of the family and hence we cannot always be sure that the incidence is really as exceptional as it appears. Furthermore, they have, of course, the defect of depending only on family traditions and belief as to the cause of death. Also, we must consider that in family records with a high incidence of cancer, no matter how high the proportion is, the possibility that the heaping up of cases in these families may depend on chance cannot be evaded. Granted that of the entire population past forty, about 10 per cent will have cancer, the laws of probability would determine the occurrence of occasional families in which a high proportion of cancer cases would occur if heredity had no influence at all.

But when we find families that have many cases of a certain sort of tumor which is not common, or a tendency to frequent location of a certain tumor in a certain place, the hypothesis of coincidence becomes inadequate. The retinal glioma families afford outstanding examples of such unquestionable hereditary influence. Glioma of the retina is a rare neoplasm, which is remarkable in that it often occurs in infants or even at birth. Its familial occurrence is equally striking, and we have numerous records of families in which several individuals have been destroyed by this growth. Another tumor of markedly familial occurrence is multiple neurofibromatosis, which often affects many members of a family through many generations. Another form of benign neoplasm with an extremely marked hereditary character is multiple cartilaginous exostosis. Here the behavior suggests a Mendelian dominant character. Multiple subcutaneous lipomatosis also appears as a familial condition, occurring, in some families at least, in a way to suggest a sex-linked recessive. Multiple benign cystic epithelioma is another growth that has a distinctly familial distribution. Adenomatous polyposis of the large intestine, with a marked tendency to carcinomatous transformation, often exhibits a familial occurrence. Melanotic neoplasms of the choroid coat of the eye, themselves rare tumors, have in several instances attacked several members of the same family. Davenport has reported one family in which this disease was observed in four generations.

Another item of evidence is furnished by those cases in which identical tumors occur in identical twins. Several such cases have been reported. I have personal knowledge of two pairs of identical twins, all four dying of malignant tumors arising in the testicle. In one pair of twins the tumors appeared within a few months, but in the other pair the interval was of a few years. But in each pair the corresponding testicle was affected and the histology of the tumors was the same. Particularly striking is the report of Leavitt of the occurrence of identical cerebellar tumors in identical twins, one at six and one-half and the other at eight and one-half years. It must be admitted, however, that the evidence furnished by tumors in identical twins is not large, for relatively few such cases have been reported, and we have no information as to how frequently it happens that one of a pair of identical twins has a neoplasm *without* a similar tumor in the other. But since many different sorts of tumors have been found duplicating one another in the correspond-

ing sites in both of pairs of twins, they at least furnish evidence that the site of tumor occurrence may be determined by heredity, as do also the families with adenomatous and cancerous growths in the colon.

Satisfactory evidence in respect to most inheritable qualities is usually difficult to get from human material because of the fundamental facts that the human life cycle is long, the families small, the matings unselected and perhaps uncertain. We, therefore, are forced to seek for evidence from the domestic and laboratory animals, which offer larger and better-controlled genetic material.

Assuming that the evidence in respect to both the genetics and the neoplasms of the laboratory animals may be utilized in studying the influence of heredity on the occurrence of cancer in man, we turn to that evidence and find that it unequivocally indicates that there is such an influence.

As pointed out previously, comparative pathology strongly indicates an influence of heredity on the occurrence of cancer, in that certain types of tumors occur more commonly in one species than in another. Possibly the recognized susceptibility of Negro women to fibroid tumors in the uterus and the apparent low incidence of breast cancer in Japanese women and of uterine cancers in Jewish women may depend on hereditary factors.

The experimental studies on the occurrence of spontaneous tumors in mice reported from the Otho S. A. Sprague Memorial Institute by Maud Slye differ from those reported by others, not only in the period of time and magnitude of the material, since over 100,000 pedigreed mice have been closely studied more than twenty years, but in the fact that not one of these mice has been subjected to any artificial influences that might modify its life, which is prolonged by careful hygiene as much as possible.

Miss Slye has unquestionably shown more conclusively than had previously been done that heredity is an essentially important factor in determining whether cancer will or will not appear under ordinary conditions of life, and in particular she has demonstrated that heredity determines in large measure, perhaps entirely, in what site and with what type of structure cancer will appear. For example, in certain strains sarcoma is very common; in others it is seen rarely or never. In some strains one seldom sees any form of malignancy except mammary-gland cancer. Miss Slye has developed one strain of mice whose inbred and hybrid derivatives have yielded

more than 100 primary liver tumors, although in all the other mice examined postmortem in her laboratory not a single liver tumor has been found and only two other cases have been reported from the thousands of mice examined in other laboratories. Another strain has yielded a considerable number of tumors of the testis, although not a single case has ever been reported from other laboratories. A single small strain of mice has yielded numerous primary tumors of the thyroid, although no thyroid tumors have ever been reported in mice outside of Miss Slye's laboratory.

Such study as others have made of the influence of heredity on spontaneous cancer serves to corroborate at least the fundamental point that heredity is a most important factor in determining the absence or occurrence of cancer. For example, in the Crocker Laboratory at Columbia University numerous experiments have been carried out with rats infested with a certain tapeworm which passes its encysted stage in the liver. In some of the infested rats sarcoma develops in the liver as a result of the irritation or growth stimulation. It has been found that different strains of rats differ greatly in their tendency to develop sarcoma in response to this common stimulation of the tissues, some giving a high percentage and some a low percentage of positive results. Wood has reported that when rats that developed sarcoma were bred together much higher rates were obtained in the offspring, such families sometimes giving 100 per cent of positive results. From the same laboratory there has been reported a large number of cases of spontaneous tumors of the thymus occurring only in a single strain of rats, while certain other tumors predominated in other strains independently of known stimulating agencies.

In view of all the experimental evidence and the absence of any experimental evidence that contradicts it, the conclusion seems inevitable that the incidence, character, location and behavior of tumors depend, to some extent at least, on the inherited qualities of the animal and of its tissues. This being granted, the next question is, How does heredity determine susceptibility or resistance to tumor formation?

Apparently this may be answered as follows: Tumor formation is the result of stimulation of the tissues to growth, the stimuli being of various sorts and nonspecific. Some stimuli produce marked proliferative effects in proportion to the retrogressive effects, and such stimuli are particularly capable of leading to neoplastic prolif-

eration, *e. g.*, roentgen rays, coal-tar. The same amount of stimulation does not produce equal amounts of proliferative reaction in all individuals, even when of the same species; *e. g.*, Negroes are more likely to develop excessive amounts of connective-tissue growth (keloids) in response to cutaneous injuries than are white men and some white men develop more scar tissue than others from similar wounds. Not all roentgen-ray workers develop the same degree of hyperkeratosis from the same amounts of exposure and some develop roentgen-ray cancer much sooner than others. Hence there are individual variations in both amount and character of proliferative reaction to a common stimulus, and these variations undoubtedly rest on the hereditary basis, in part if not wholly.

Evidently, then, heredity may determine whether the proliferative reaction that follows injury assumes a neoplastic character or not, just as the Crocker Laboratory rats do or do not develop sarcoma in the liver about the encysted tænia, according to their ancestry. This has been pointed out especially in connection with the study of the lung tumors in Miss Slye's mice, which show that mice with cancer heredity react to nonspecific inflammatory conditions in the lungs more often with excessive proliferation which leads to malignancy than do mice of noncancerous ancestry. All mice of tumor age have suffered more or less from inflammatory conditions in the lungs, with proliferative reactions. In some of the mice of cancer ancestry the proliferation assumes a definitely neoplastic character. On the other hand, mice not of cancer ancestry, subjected to corresponding lung injury, very rarely, if ever, react with proliferation to a degree even suggesting tumor formation. Since the susceptible animals are not born with cancer, but develop it only later in response to certain environmental conditions, it is evident that only the potentiality, or lack of resistance, is inherited.

Taking all the evidence, whether from clinical experience or laboratory observation, it seems impossible to escape the conclusion that the occurrence of cancer both in animals and in man is influenced by heredity, and it seems most probable that the hereditary influence is an extremely important one. Admittedly the occurrence of cancer depends often upon the existence of a chronic irritation, whether physical or chemical. How readily such irritation will lead to the development of cancer apparently depends largely upon the inherited capacity or lack of capacity to prevent the reactive proliferation from passing from normal to neoplastic char-

acter. The fact that many, but not all, mice of unselected ancestry may develop cancer after painting with tar, and that the duration and amount of this irritation required to produce cancer vary greatly in different animals, indicates the existence of different individual capacities to resist cancerogenic stimuli. The fact that so many of the pioneer x-ray workers have developed cancer indicates the possibility that an adequate stimulation may overcome the resistance of almost any human being. On the other hand, the fact that in cancer families, and in experimental cancer strains, the ordinary amount of stimulation common to all the species leads to cancer in most members of the family or strain, whereas a similar amount of stimulation rarely produces similar tumors in the rest of the species, indicates the possibility of inheritance of minimal capacity to resist the appearance of the malignant type of cell proliferation. For example, no particular irritation seems to be required to cause retinal gliomas to form in the young children of glioma families. And what particular irritation could there have been in the roof of the fourth ventricle of each of the identical twins, described by Leavitt, who developed tumors at this well-protected point? A particularly illuminating history is given by Warthin of four brothers with cancer ancestry on both sides of the family. Three, who were smokers, died in middle life of cancer of the lip; the fourth did not smoke, but nevertheless developed cancer of the lip at a much later age than his brothers. Here the susceptibility to cancer of the lip was present in all four, but the greater irritation of the lip in three caused an earlier development of the malignancy.

At the present time it seems safe to maintain that the existence of an hereditary influence on the susceptibility and resistance to cancer has been established both for man and animals. The exact mechanism of the hereditary influence has yet to be determined. The evidence offered by human material is conflicting, and inadequate both in amount and character to permit of satisfactory analysis. The studies so far made with spontaneous tumors occurring in laboratory animals show the possibility of modifying the occurrence of cancer in these animals in marked degree, and of determining by experimental genetics the site and character of the tumors that will arise. Therefore, we may reasonably look forward to the eventual finding of a definite explanation of the genetic mechanism which determines susceptibility and resistance to human cancer.

WHAT KOCH MEANT TO TUBERCULOSIS*

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VERY early on the morning of April 3, 1882, a very much excited elderly man, with graying hair and the sideburns typical of the practitioner of those days, rushed into the bedroom of a much younger man, a great student, a reader into the wee hours of the night, and awakened him, waving the morning newspaper and shouting: "I knew it! I knew it! I knew it!" In this way Austin Flint, a firm believer in the contagiousness of phthisis, informed Prof. William H. Welch of the discovery of the tubercle bacillus. Ten days before Robert Koch had traced the life history of this germ before the Berlin Physiological Society, a small but influential society, where frank discussion was the rule and ambition was often none too gently treated. That night, when the speaker finished reading his communication, all eyes turned on the great Virchow, who had combatted the idea that caseation might be due to tuberculosis; but he slumped in his chair, manifested no desire to speak, for criticism he had none, and taking his hat he left the room in silence. "That evening remains graven in my memory," wrote Ehrlich later, "as the most majestic scientific event in which I have ever participated." On that same morning when Flint awakened Welch, my earliest medical recollection takes me back to a dining room where I heard the family discuss and read in the *Sun* paper the headlines of the cablegram describing how the cause of consumption had at last been discovered.

To translate ourselves fifty years backward into that period of the world when Koch made this discovery is difficult but interesting and, I might add, necessary, if we wish to comprehend its significance at that time. There is no time tonight to recall the political conditions and I shall confine myself to an attempt to give you some conception of the state of hygiene and something of the ideas concerning tuberculosis in existence at that time.

* Read at the meeting of the Section on Medical History of the College of Physicians of Philadelphia, March 14, 1932.

The germs of anthrax, malignant edema, gonorrhea, relapsing fever, leprosy, pneumonia, typhoid fever, glanders and staphylococci, streptococci and the malarial plasmodium had all been discovered. You will recall that Joseph Leidy of Philadelphia, in 1849, had placed these germs among the plants, and that Prof. J. K. Mitchell, father of Weir Mitchell, had, in 1849, been one of those who believed in the germ theory of disease. But the germ theory at this time held that the germ was not superimposed upon the patient and independent of him; it was something belonging to him and borrowing from him a sort of pathological vitality and ability to transport it elsewhere. The idea of a living virus, able to be cultivated and modified outside the body, was to be new with Pasteur.

No work relative to obtaining a good milk supply had yet been undertaken. That flies could transport anthrax germs had been shown, but their relation to other diseases had not been suggested. Filtration of water was used by 30,000 persons in America in 1880, and today the number must be nearly 50,000,00. The first hygienic institute, that of Pettenkofer, in Munich, opened in 1878; the first in America in 1884, at Bellevue Hospital, where Welch had inaugurated his bacteriological laboratory in 1878, while Prudden began his at the College of Physicians and Surgeons in New York a year later. Both had been pupils of Koch. The bogie of carbonic acid gas in regard to ventilation was still in the minds of many sanitarians at this time and most houses were heated by stoves and hot-air furnaces.

It is Koch's work on tuberculosis that interests us chiefly tonight, and for us to grasp its significance we must know something of the ideas in existence at that time concerning tuberculosis. "Nothing further than contagion is necessary for the origin of phthisis," wrote Jerome Fracastor, the founder of the germ theory of disease, in 1546, "which may attack a person who has never had catarrh, who has not ruptured a bloodvessel or has an ulcer about the chest or who has not had a pleurisy or any other malady, but has been perfectly well. He may contract it by habitual intercourse or a life in common with a phthisical patient, or by fomites (contaminated objects). For it is astonishing how tenacious and for what a long time the virus will live in fomites; we have seen the clothes which a consumptive had worn to give the contagion after two years; indeed, the bedrooms, the beds and the flats (?) in which

the patients have died may do the same." The Italians and the Spanish were both convinced in the eighteenth century of the contagiousness of phthisis and required that bed and belongings of any one dying of the disease should be burnt. But in the North such views did not hold. You may recall that Sauvages, in France, described 20 varieties of phthisis, and Morton, in England, 16, but that Laennec preached the unity of phthisis and believed that all the changes in the lungs in pulmonary tuberculosis arose from the tubercle. Louis upheld Laennec. Klencke later, in 1843, showed that tuberculosis might be transmitted by cows' milk. But many combatted these ideas, and among them none was more commanding than the great Virchow, who held that tubercles had no connection with the cheesy matter so common in pulmonary tuberculosis (caseous tuberculosis), and that the cheesy degeneration might follow a variety of causes. Niemeyer even went so far as to say that the worst thing that could happen to a consumptive was that he might become tuberculous. Heredity at this time was considered the most important factor in tuberculosis, and in such unfortunates cold, humidity, dust, among other things, might bring about the disease. But no germ had yet been proved to cause the disease though many accepted such a view. In a few years, however (1865 and 1868), Villemin proved that "tuberculosis is the effect of a specific causal agent, of a virus," and that dried sputum was important as a source of the disease. His book is a model of scientific writing, but none is so difficult to convince as the self-satisfied and even the definite proof of Villemin's contention brought forward by Chauveau, Edwin Klebs, Gerlach and Cohnheim failed to settle the problem for these doubters such as Colin, Chatin, Virchow. Cohnheim predicted, however, that soon some one would find "tubercle particles" in the tubercles and in the cheesy matter. Stimulated by this, many set out at once to work on the problem, and, in 1877, Klebs announced that he had found the cause of tuberculosis, and Aufrecht did the same; Toussaint in 1881 and Baumgarten in 1882 made a similar claim. While it is probable that Baumgarten did really see the tubercle bacillus, he was unable to stain them or to produce convincing proof of his belief.

You may recall that Koch began his bacteriological work in a small curtained-off portion of his office while in private practice. Here he isolated for the first time a pure culture of a bacillus, the anthrax bacillus; here he first made use of a solid transparent

gelatin culture medium. You will remember that he left Wollstein for Berlin in 1880, and in the fall of 1881 began work that consumed much of the time he was allowed to spend at home until his death, in 1910. That is the study of tuberculosis. At this time Bodington had passed into forgetfulness and Brehmer and Dettweiler had ushered the treatment of pulmonary tuberculosis into the sanatorium period. There was no sanatorium as yet in America, but shortly Trudeau was to lay the foundations of his on a bleak hillside. As the question of contagiousness was not settled, no sanitarian dared suggest any sanitary regulations concerning tuberculosis; there was no fumigation, no sterilization, no spitting ordinances, no tuberculin for diagnosis. Brehmer was still exercising the "small hearts" of his patients, and Dettweiler was advocating rest in the open. All was still empirical. Forlanini had begun to think of artificial pneumothorax, but had not yet dared to practise it. Diagnosis of pulmonary tuberculosis had not advanced much beyond the state that Louis left it in the early part of the century. Epidemiology and prophylaxis were unborn.

After Koch left Wollstein and went to Berlin to begin work in the Imperial Board of Health, where the study of bacteriology had for some time been neglected, there began to appear communications from this body in the form of large folio volumes, in the second of which, published in 1884, was a more extended article on the etiology of tuberculosis, by Koch, filling 88 pages and accompanied by 53 excellent chromolithographs portraying the cultures and pathological sections and specimens from man and animal. No work was ever more complete, no work ever had less adverse criticism and no work has ever stood the acid test of time more successfully. It is regrettable that we have not time to present to you a full review; but I cannot pass it by without a brief mention of some of the important facts it contains. The nature of the disease is, Koch states, still in doubt and clinical observation has been unable to solve it. Its infectiousness has been demonstrated, but the infectious agent remains unknown, whether living or inorganic, whether capable of growth or inert. The ordinary methods of search for the organism failed until the stain, methylene blue, was rendered alkaline and then in smears and in sections, decolorized and counterstained later, showed fine, thin, blue rods. Since 1882 better methods have been devised and Koch gladly accepted and used them. The bacillus resisted decolorization by acid or alcohol.

Having found a germ, it became necessary to see if it were present in all forms of tuberculous lesions, in miliary and pulmonary tuberculosis and all other forms in man and animal. They were. "From these facts," Koch writes, "viz., that tubercle bacilli are always present in tuberculosis and occur nowhere else, that they precede, both as to time and place, all the peculiar pathological reaction changes, that their number, appearance and disappearance are in direct relation with the course of the disease—the conclusion may be drawn with great probability that the tubercle bacillus is not a chance accompaniment of tuberculosis, but stands in the position of the direct cause of the disease."

He had difficulty but finally succeeded in growing the bacillus for many generations outside the body. I can see the Master as morning after morning on his arrival at the laboratory he went immediately to the thermostat. I can picture the grim, determined look on his face as day after day, for seven days, for eight days, for nine days, he saw nothing. I have no difficulty in conjuring up the look of pleasure that spread over his face as at last on the tenth day he saw revealed under his lens little scales and white spots. For inoculation he used bits of tuberculous tissue with bacilli in them and pure cultures and was uniformly successful, while the controls with blood serum were negative. So he concludes that the tubercle bacillus is the cause of tuberculosis.

In his article Koch then proceeds to discuss the relation of the bacillus to the etiology of tuberculosis. In the first place the germ cannot grow outside the body of an animal, for it requires a constant temperature of at least 30° C. No facts support the view advanced by some that the tubercle bacillus can assume other forms outside the body, nor is there any evidence as yet that after two years the bacillus loses any of its virulence. Later he modified this view. No doubt the tubercle bacilli did originate from another species of bacteria, but now they appear stable. So all tuberculosis comes from preëxisting tuberculosis and the most common forms are phthisis and tuberculosis of domestic animals. He mentions the danger of sputum in fine particles and dried sputum in the form of dust, which he thinks is more important. Handkerchiefs are especially dangerous. He notes how long the tubercle bacillus is in developing and thinks this the reason why so many escape infection, for the bronchial cilia sweep out the bacilli before they can develop unless, as in measles, the respiratory tract is denuded for a

time of its protecting epithelium or a secretion is produced difficult to dislodge and suitable as a nidus for the tubercle bacillus. Adhesions of the lungs may bring about a similar condition. So not only the bacillus but some favoring condition is necessary for infection in man. He fails to mention the tonsils as a port of entry in tuberculous cervical adenitis.

The danger from bovines is concerned chiefly with milk and meat; but he does not stress the danger and thinks the only dangerous cow is the one with a tuberculous udder. He doubts whether a single case in the human subject will ever be referred unquestionably to the partaking of the flesh or milk of tuberculous animals. However, from a hygienic point of view, the same precautions must be taken against infection with the bovine tubercle bacillus as against the human form.

There is, he notes, a striking difference between tuberculosis in children and in adults, and apparently a predisposition to infection in some families, due in some instances no doubt to increased opportunities for infection. A tendency to catarrhal affections of the respiratory organs or poor development of the thorax might explain a predisposition in some, but others cannot be accounted for. From a study of autopsies, it is found that tuberculous foci may reach a fair size, then contract, cicatrize and heal. He interprets this as indicating that what was at first invasion a favorable soil, later becomes changed into a bad soil, which prevents growth and so favors healing. What brings this about he leaves to the future to determine. He notes how rare is uterine infection and explains what may be inherited is not the germ but a soil favorable to its growth.

In summarizing the value of his work, he states that he brings nothing new to the etiology of tuberculosis, for Cohnheim had already recognized the disease as infectious and sketched the etiology. He has, however, met all objections to such a view and furnished a criterion which will determine what is and what is not tuberculosis, and which, of course, aids diagnosis. He lays particular stress upon prophylactic measures, as from the experiments he has performed there does not appear any great prospect of finding a therapeutic means of dealing with the parasites in the body. Prophylactic measures, before introduction, must be carefully considered or otherwise unavoidable disturbances and other disadvantages will neutralize the benefit to be obtained.

So ends a masterpiece of medical literature. One of the most remarkable things in medical history is how little of moment has been added to our knowledge of the tubercle bacillus since the appearance of this contribution. With it Koch probably reached the zenith of his fame, of his work.

Koch's influence on medicine in America, as elsewhere, has been profound. Sternberg, later surgeon-general of the U. S. Army, demonstrated and photographed in 1882 at Fort Mason in California, one of the first in America, the tubercle bacillus. The Saranac Laboratory for the Study of Tuberculosis, founded by Trudeau in 1894, was the direct outcome of Koch's monograph. Welch of Baltimore, Prudden of New York, who taught Trudeau how to stain the bacillus, Ernst of Harvard, all worked directly under Koch in Berlin. Victor Vaughan, Novy, Biggs, Ford and others studied in the Institute of which Koch was the head.

It is interesting to recall how slow some good clinicians were in accepting the importance of the tubercle bacillus in diagnosis. Dr. Trudeau told me many years ago of a young Harvard student who, suspected of having pulmonary tuberculosis, was sent to him for diagnosis. Not long before this Trudeau had climbed the narrow steps to Prudden's little laboratory in New York to learn how to stain the tubercle bacillus. He was led to do this from his study of Koch's original paper of 1884 which Mr. Charles M. Lea, of the medical publishing house of Philadelphia, had been good enough to have had translated for him, an act which was really at the bottom of the foundation of the Saranac Laboratory, as I mentioned. In the sputum of this young Harvard student he found many of the slender rods so well described by Koch, though in the patient's chest he could detect no evidence of disease. The family were not satisfied and took the boy to a leading specialist in New York, Dr. Alfred L. Loomis, himself a sufferer from the disease tuberculosis, and later the first to demonstrate the presence of tuberculosis in apparently normal lymph glands (1890). Loomis heard nothing abnormal in the patient's chest, pooh-poohed the idea of tuberculosis and allowed the boy to return to Harvard. In a year or two he relapsed and eventually died. Few such instances were necessary before the value of the tubercle bacillus in diagnosis was fully grasped. But as soon as this was fully appreciated, many concluded that every case of pulmonary tuberculosis must have tubercle bacilli in the sputum, which Koch's paper clearly led them to infer,

and, on the contrary, if the tubercle bacilli were not found the patient could not be suffering at that time from tuberculosis. It required many years to correct this error and, when corrected, many have gone too far and now attach too little significance in diagnosis to the presence or absence of the tubercle bacillus in sputum.

In spite of Koch's advice that chemotherapy would offer little aid in any attack on the tubercle bacillus in the body, a feverish search for some such substance began at once. The germ had been found and, now that anthrax and hydrophobia rabies had been conquered, tuberculosis must soon succumb. Innumerable substances were given by mouth, by injection and by inhalation. Hydrofluoric acid, for example, was inhaled until the mirror and all the windowpanes were etched and ruined—and still the tubercle bacilli went on the even tenor of their way in lung or other tissue.

In 1890 Koch announced the discovery of a specific remedy for tuberculosis that would cure the diseases in skin tuberculosis (lupus), in bone and in early cases of pulmonary tuberculosis; more severe cases might be benefited. The world stood aghast and delighted. The nature of the remedy he did not divulge when he made his communication to the International Congress on August 4, 1890, and, in fact, only when it became highly probable that others would announce its nature did Koch, in the following January, state that it was glycerin extract of the tubercle bacillus, known at first as "Koch's lymph" and later named by Bujwid (1891) "tuberculin." "No medical discovery," wrote Trudeau, "has ever produced such a sensation as the startling announcement coming from the great scientist who had discovered the tubercle bacillus. Medical men from all over the civilized world flocked to Berlin to witness the effect of the remedy and to obtain some for their use if possible. A thousand dollars were freely offered for a bottle of tuberculin, which contained about a teaspoonful, and the substance could not be procured at that price from those who had been fortunate enough to procure some. Crowds paraded the streets in Berlin chanting rhymes in Koch's honor and shouting his name." Biggs, Loomis and many others took the first boat for Europe. Many patients in Saranac Lake packed their trunks and left for New York to be near the docks so as to lose none of the few precious days that remained to them. And then came the sad realization of the truth! Overdosage and disastrous reactions occurred, followed apparently, in a few far-advanced cases, by death. Virchow said

it spread the disease, but this was doubtful. Great resentment arose and much abuse was heaped on Koch, who through it all remained silent and at work. In November, 1890, you may recall that Trudeau published the failure of his attempts to immunize guinea-pigs and rabbits by the injections of filtered and sterilized liquid cultures, really unconcentrated tuberculin. In 1892 Kitasato, under Koch's direction, published details of experiments upon guinea-pigs which revealed that Koch had obtained only partial immunization, which Trudeau and others had recorded at the same time. It was rumored that high authorities (even Emperor William some whispered) had forced Koch to make a premature announcement; but again in April, 1897, he writes of a new tuberculin, "T.R.," tuberculin residuum, capable of protecting guinea-pigs against subsequent virulent inoculations and of producing attempts at the formation of scar tissue, provided the treatment be begun within a short time of infection (two weeks), stating that "anything better of that kind cannot be made, and whatever is to be gained by tubercle cultures must be obtained by means of this preparation."

Again, as Trudeau points out, he fails to bring evidence of his claim. "I succeeded," Koch wrote, "in rendering a large number of guinea-pigs completely immune, so that they submitted to repeated inoculations with various cultures without being infected." It is difficult for us now to see how Koch could have been so misled, but he gives no protocols to uphold such assertions, and, indeed, we find him again in print (in November, 1901) advocating the use of still another tuberculin, "B.E.," bacillary emulsion, a simple emulsion of crushed, ground-up tubercle bacilli containing, as was found later, a few living tubercle bacilli, and urging serum agglutination as the best means of regulating the individual dose and suggesting its intravenous use.

Koch's great eagerness to make practical use of his discoveries, as W. H. Park notes, played a prominent part in all of his work. During the earlier years it was of immense benefit, but later in his life it tended to lead him astray, possibly to entice him to draw conclusions based on insufficient evidence. This unfortunate eagerness, so little manifested in the earlier years of his life have led some to state that with the publication of his article on the discovery of tubercle bacillus he reached the pinnacle of his career. They are probably correct.

It has long been realized that in medical science carefully recorded,

accurate observations make any communication of lasting value, though the explanation offered may quickly be cast aside. This has been true of what we today call the Koch reaction or phenomenon. When a dose of tubercle bacilli is made into the skin, or just under it, in a normal guinea-pig, little is seen until the tenth to the thirteenth day following, and then gradually an ulcer forms, which persists until the death of the animal. Now Koch noted that if he reinjected a tuberculous guinea-pig, there was a much more rapid, a more violent reaction at the site of injection, followed by the formation of an ulcer which, as a rule, healed quickly and permanently. This is the Koch phenomenon, the significance of which led Koch to discover tuberculin. Indeed, until a young pediatrician, a child's specialist, Clemens von Pirquet, working in Vienna, first with vaccination, later with tuberculin, called attention to it, it was never fully comprehended.

Now Koch had also called attention to the fact that if a very small quantity of tuberculin, $\frac{2}{10\,000}$ teaspoonful, were injected under their skin, many patients with tuberculosis would have within eight to twenty-four hours a feeling as if they had grippe and run an elevated temperature for a few hours and show some reaction at the site of their disease. A healthy man, like himself, Koch said, would not react in this way even when ten times this dose was injected. This subcutaneous tuberculin reaction was widely used but was criticized by some observers as dangerous and many ceased to use it on this account, though in Saranac Lake we met with few if any unpleasant permanent results, and we continued it use under certain conditions. Virchow, you may recall, stated the injections of tuberculin under the skin mobilized the tubercle bacilli and thus caused a spread of the disease, which we could not demonstrate on animals. Pirquet found that injection of the tuberculin into the skin removed all such objections. Later it was found that all tissues of the body reacted more or less in this way and many variations of the test were described. It was difficult to estimate how great has been the value of these tests in the anti-tuberculosis campaign.

At the British Congress on Tuberculosis, held in London in 1901, Koch called attention to the danger of attacking all pestilences alike and recalled that leprosy, very similar to tuberculosis, was being controlled by the isolation of only certain cases. Pulmonary tuberculosis, he thought, was due to air-borne infection. In his

first communication on tuberculosis Koch expressed himself regarding the identity of human and bovine tuberculosis with some reserve and had to leave the question undecided, but now he felt that they differed and human tuberculosis could not be transmitted to cattle, for in 19 cattle injected with the human bacillus none developed tuberculosis. The more important question, whether man is at all susceptible to the bovine bacillus, is not yet and will not be for some time absolutely decided; but the danger from milk and butter and flesh of tuberculous cattle is hardly any greater than that from heredity. "I do not deem it advisable to take any measures against it," he stated. The improvement of the domestic condition of the poor will lead to a considerable diminution of the disease. He strongly advocated the erection of special hospitals for the advanced cases, and even if only a fraction were thus housed it would lessen infection. Obligatory notification was, he thought, very valuable, as well as disinfection and education about infection. Sanatoria will never render these measures superfluous. It is of interest to recall that Ravenel, working at that time with Pearson in Pennsylvania, opposed the view that the bovine tubercle bacillus could not infect man, but had made, of course, no study of the bacilli in pulmonary tuberculosis.

At the Washington Tuberculosis Congress, in 1908, Koch recalled that in London he stated that human and bovine tubercle bacilli are different: human beings may be infected with bovine bacilli but serious disease rarely develops, and that preventive measures should be directed primarily against the human bacillus. He stated that the work of Theobald Smith, on the differences between the human and bovine, led him to take up this work. He proceeded to lay down laws to be followed to prove such views, and stated that the only experiments that met these conditions were those conducted at the Imperial Board of Health in Berlin; those of the British Commission do not. He refused to accept any intermediate type of bacillus. "To my knowledge, the bacilli of the human type have never been found demonstrated in cattle." "Up to date, in no case of pulmonary tuberculosis," writes Koch, "has the tubercle bacillus of the bovine type been definitely demonstrated." They, however, have been found in the cervical lymph nodes and in the intestinal tract. At the same Congress there was held a meeting *in camera* at which I was lucky enough to be present. Dr. Theobald Smith can tell of what transpired, but I was im-

pressed by the rather gruff, somewhat overbearing attitude of Koch, which contrasted rather unpleasantly with the gracious manner of Arloing, of Lyons.

The endeavor to present to you tonight the ideas prevailing about tuberculosis at the time Koch made his memorable discovery will help you to infer what addition his work has made to our present conception of tuberculosis. He stated, you will recall, that Cohnheim had already conceived the idea that it was an infectious and contagious disease and had sketched its etiology. But few accepted such ideas until Koch brought the definite proof. From that time onward direct heredity of the germ has played only minor part, for the bacillus has only in rare instances been found in the infant at birth. This Koch foresaw. All our ideas of the distribution of the bacillus in or outside the body of man or animals have been developed since the bacillus was discovered. At first it appeared as if the problem of the modes of infection had been solved by Koch when he described conveyance of the bacillus by dust and intimated that droplets might play some part, a problem much worked over by his assistant Flügge. It must be acknowledged, however, that this problem is still far from settled, if we but recall the controversy over whether pulmonary tuberculosis starts from the primary or from a secondary infection, and whether that comes from without or from within, in childhood, adolescence or adult age, by inhalation or by other portals of entry. The bacilli must often gain entrance into the blood; but still today we have great difficulty in discovering them there.

The pathology of tuberculosis was placed upon a firm basis by this discovery. The connection of the tubercle with tuberculosis, starting with Sylvius, was held and modified by Manget, Morton, Stark, Reid, Baillie, Bayle to Laennec, Louis and Rokitansky. Virchow opposed this conception; and only by such a demonstration as Koch gave could such authority be overcome. Where the tubercle bacillus could be shown to be the only or predominating organism, there the pathological change must be tuberculous. The whole subject had to be reworked, though much had already been done by Koch himself.

The importance of the bacillus in clinical diagnosis has already been mentioned. Koch stated that careful research never failed to disclose it in the sputum of cases of pulmonary tuberculosis. Today we insist upon search for the bacillus in all cases, but recog-

nize also, as did Koch, that in some discharges, in some pus from tuberculous lesions, it is difficult if not impossible to find the bacillus when inoculation of the discharge produces tuberculosis in the animal. The nearly constant presence of the bacillus led for a time to the postponement of the diagnosis until it could be found, a procedure which deprived some patients of a chance to recover. This is now no longer followed. Many methods of searching for the bacillus in the feces, urine, pleural fluid, stomach contents and other secretions have developed in due time. The discovery of the filterable forms may yet prove Koch's contention that the bacillus is always present in the sputum in pulmonary tuberculosis.

The importance of the bacillus in the treatment of tuberculosis is shown in many ways. In the first place, it still remains the only absolute proof of the presence of tuberculosis. In some rather rare instances it alone makes the diagnosis of pulmonary tuberculosis positive. In these and other doubtful cases it emphasizes the importance at once of rigid treatment. We infer when tubercle bacilli are present in the sputum that ulceration into a bronchus has occurred and *per contra*, when they disappear, that ulceration has healed. Some insist upon the inoculation of the sputum into a guinea-pig, so important do they consider this point. Following an acute bronchitis, bacilli may reappear for a time. The bacillus has served as a starting-point in chemotherapy for Koch and many after him; but so far, as Koch predicted, no efficient treatment has arisen. Many, however, still pursue this bacillary chimera, as some would term it. I shall not be surprised if, by chance, some fortunate worker may happen upon such a substance that works very differently in the body than it does in the test-tube.

Probably in no branch of tuberculosis study has the discovery of the bacillus exerted so fruitful an influence as in prophylaxis of the disease. At this time one-seventh of all deaths were due to tuberculosis. In 1882 the mortality-rate in cities of the United States was 326 per 100,000, while in Philadelphia it reached 318, in San Francisco 323 and in New Orleans 396; while today in the United States it has fallen below 80, reaching in New York, in 1930, 69, and in Philadelphia 71. Koch stated that the bacilli in the sputum were the most important factor in the spread of tuberculosis, and later, as you know, denied the importance of milk from tuberculous cows. It is difficult to follow blindly the views of the Master in regard to milk; for, since pasteurization has

been widely employed, tuberculosis in the early ages has greatly decreased. Whether this is *post hoc* or *propter hoc* is not yet definitely settled, though I incline to believe that pasteurization has exerted much influence. Not a direct but an indirect attack upon the disease, carefully planned, as Koch suggested, has brought this about. Seizing upon these facts, Biggs forced upon the city of New York most efficient measures against the disease. Flick, standing practically alone, forced eventually upon an undivided, doubting, medical profession, gathered together in this very College to oppose his views on house infection, laws based upon sound ideas. Cleanliness, disinfection, proper lighting, sufficient ventilation, institutional care for advanced cases (long in use in Philadelphia at Chestnut Hill Hospital), proper methods of dusting, dispensaries, sanatoria, the proper disposal of sputum, the covering of the mouth when coughing and a thousand and one other measures have brought the mortality from tuberculosis, in spite of war, in spite of depression, to such a figure that, thanks to Robert Koch, before many decades tuberculosis will be considered to be one more of the great infectious and contagious diseases that man has conquered.

GOETHE AND HIS THEORY OF COLOR*

By BURTON CHANCE, M.D.

(Abstract)

Dr. Burton Chance read an extensive account of the controversy which agitated the scientific and philosophical world over the "theory" advanced by Goethe, in 1791, for the solution of the mysteries of color vision and color mixing, and later, in his famous *Farbenlehre*, published in 1810, which controversy continued with undiminished acerbity for many years after the great poet's death.

Goethe would not accept Newton's teaching, that white light is compounded of all the colors found in nature; that by means of a prism the passage of rays of light through it the rays are broken and, when collected on a screen, they are arranged in several colors in the order so well known as the spectral, and, accordingly, from Newton's experiments in 1666, white light was believed to be a mixture of all colors in certain definite proportions. Newton further explained that the perception of the colors of the rays of light depends entirely upon the result of the action of the rays upon the retina; that the rays are not red, or some other color, but they produce the sensation of color.

Goethe, from his studies in painting while still a youth, became interested in the mixing of colors and sought to comprehend the teachings of Newton which he had learned during his University days. In his experiments with prisms he was disappointed in not obtaining the response which Newton had so readily obtained in his darkened room in Cambridge. He had expected to see, as he looked out his window through the prism, the spectral colors reflected on the white wall, but he could obtain the sequence when the clear expanse was broken by a dark object. This he believed vitiated Newton's doctrine, and he set about to combat what he regarded as fallacious in it, ever mindful that Sir Isaac too had expressly noticed the effects of colored boundaries or other limita-

* Read at the meeting of the Section on Medical History of the College of Physicians of Philadelphia, April 11, 1932.

tions to the bright reflexions. He advanced a theory of his own by declaring that light is homogeneous and the simplest matter known; it is not compounded of colored lights; colored lights are darker than colorless light, brightness cannot be compounded by darkness. Blue and yellow are the only pure colors, together they make red; green and purple are mixed colors, all other known colors are gradations of these and are not pure. Colorless light cannot be produced out of colored lights nor white from colored pigments. The colors arise out of a modification of the light; colors are excited in the light. Yellow is light seen through a thickened medium, and out of these all color is derived.

His great *Farbenlehre* is in two volumes of 1510 pages, in which he compiled the known history of color as observed by the artist and the physiologist. He describes the colors of minerals, plants, worms, insects, fishes, birds, mammals and men. The headings of the chapters indicate the extraordinary industry of the poet, but they show an absence of that scientific definiteness which he stigmatizes as pedantry in the case of Newton. In a critical study of this portion of *Farbenlehre*, it must be borne in mind that at the time Goethe wrote it the anatomy and physiology of the eye were imperfectly known, and many of the phenomena as found by him are understood differently by us today, from our more complete investigative methods. He describes cases of certain persons who expressed difficulty in perceiving blue. He makes no reference to Dalton's publication concerning colorblindness nor, indeed, to the great doctrines promulgated by Dr. Thomas Young, which were later confirmed by Helmholtz. The remainder of *Farbenlehre* is devoted to the condemnation of Newton personally and his scientific theories.

The work excited the greatest excitement as well as the distrust of physicists and physiologists. Goethe's experiments were accurately observed and vividly described, yet his methods were altogether foreign to those commonly used in physics or in the treatment of a purely physical theme. Although usually temperate and courteous, in his analysis of Newton and his works he was passionate and contemptuous, and, in his discussions with his contemporaries, he asserted that this contradiction of himself was a contradiction between Goethe and Truth. He was lauded by poets, anatomists and princes; and others unaccustomed, as Helmholtz declared, to the accuracy of scientific investigation. Nevertheless, he was

unable to obtain the approval of the French Academy. Despite the rejection by scientists, Goethe advocated his theories to the end of his days, and left it with sure confidence in the judgment of posterity. The work is now dead and its conclusions unknown to most of those who are working in the department of science with which it is connected.

The experiments and observations he has recorded are astonishing in their variety and number, yet it is difficult to come to any conclusions regarding Goethe's real conceptions; they are beyond the understanding of most students of physics, although many of his observations have become the implements of the experimental psychologists and the student of physiological optics.

It is difficult to realize that the principal contestant in that acrimonious controversy was a man of extraordinary abilities who had ranged against him the foremost physicists and philosophers. He was himself possessed of, in matters poetic, the talent for seeing through whatever obscured reality, yet, in this scientific study he was convinced of the correctness of his own views and charged his opponents with narrowness or malice. Some perversion of principle or difference of organization between his mind and theirs prevented each from understanding the other. Goethe was essentially a poet; his mind could not, as could the scientist's, arrange the phenomena of Nature under definite conceptions. Goethe believed that Nature has no secrets from the observer and he was opposed to experimental research. He rebelled against physical theories, conceiving all things as an artistic whole. He desired Nature to reveal her secrets of her own free will; that all her mysteries might be observed by the beholder's unaided senses. He preferred the open air rather than the dingy laboratory. Goethe had no theory at all, and he was unable to explain the phenomena he observed. In his contributions to science in other branches his renown continues undiminished, but in this he failed lamentably because he did not utilize the methods necessary for the completeness of its principles. Scant reference is made today to Goethe's theory of color; doubtless, because of the futility of the theories and the bitterness of the controversy men have been led to suppress the incident in the light of Goethe's accepted greatness in other fields of philosophy and art.

APPENDIX

INTRODUCTORY NOTE TO THE SECTIONS

Almost all of the papers read before the various sections of the College are subsequently published in full in medical periodicals.

The papers listed in the following pages may be found by consulting the *Index Medicus*, provided a sufficient length of time has elapsed from the date of publication to the next succeeding quarterly number of the *Index Medicus*.—[Editor.]

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY, 1932, TO DECEMBER, 1932

JANUARY 21

A Corneal Dystrophy

Dr. J. Milton Griscom presented a case of dystrophy of the cornea in an Italian female, aged thirty-one years, who applied to the Wills Hospital on January 4, 1932, because of failing vision in the right eye.

External examination revealed the presence of numerous small, superficial, discreet white spots located in the pupillary area of the cornea of each eye, unaccompanied by any evidence of irritation. The epithelium was uniformly smooth and its reflex was unimpaired. There was some loss in corneal sensitivity. In the right eye there was an area of old choroiditis and adjacent to this an area of acute exudative choroiditis in the upper outer quadrant of the right fundus, accompanied by many large vitreous opacities. The fundus of the left eye was negative.

A slit-lamp examination of the cornea was made by Dr. Alfred Cowan, who reported as follows: "There are a number of grayish lesions varying in size up to 0.2 mm. in diameter, more or less regularly distributed over the central portion of each cornea, extending over an area about 6 mm. in diameter. The surface of the cornea in specular reflection is seen to be perfectly smooth. By direct illumination it can be seen that most of the lesions consist of a flat, granular, slightly concave surface with well-defined but irregular edges. There are also some dot opacities and some opaque streaks. With a narrow slit, the concave lesions, which are the most common, are seen to lie mostly beneath the epithelium, in Bowman's membrane or just behind it, a few extending back to about the middle of the cornea proper. The dot and streak opacities are mostly in

the substantia propria. The cornea between the opaque lesions contains very fine granules, giving the whole a slightly hazy appearance. By retroillumination the lesions are opaque. Descemets membrane is intact. There are some brown K.P. The lenses are clear, with many brown dotlike vitreous opacities in the right eye."

The case appeared to be similar to the type described by Fehr and allied to the group of corneal dystrophies first described by Groenouw.

Dr. Cowan said that in Vogt's new atlas will be found an illustration almost exactly like Dr. Griscom's case. It is classed among the nodular corneal degenerations and called superficial disseminated degeneration. Dr. Griscom brought out in his paper that different types of these corneal dystrophies are nothing more or less than different stages of development of the same process. Dr. Cowan thinks there is no doubt about this. This is shown in Vogt's atlas by illustrations of 4 cases in members of one family, two sisters and the only child of each, at the ages of five, sixteen, thirty-three and forty-two years. In the child of five years, there are shown only a few radiating lines in the cornea, not elevated, but just made out. The nodular form of this condition is probably a later stage of development. As a matter of fact, in Dr. Cowan's experience, nodules are the exception rather than the rule—more often streaks, flecks, lines are seen which are all variations of the same condition. Another point, these lesions are not necessarily limited to the anterior portion of the cornea; the condition is slowly progressive. Very rarely similar lesions have been observed in the posterior portions as well as in the anterior portions of the cornea.

Dr. Zentmayer asked Dr. Cowan whether he had noted the condition of the corneal nerves and said that one observer had stated that they are thickened. In reply, Dr. Cowan said it was not especially noted. He did not consider that increased visibility of the corneal nerves is particularly significant.

Dr. Zentmayer said that in "Contribution to Ophthalmic Science," together with Dr. Rush, he had reported cases seen in three of four generations, of which direct information could be obtained, and the greatgrandmother was said to have been similarly affected.

Bearing on the view that the various types of dystrophy described are all the same, seen at different stages, he would state that there was a considerable difference in the ages of their patients when examined, and presumably, from the statement of the patients, also in the length of time they had been affected; but in all the lesions were practically the same.

Posterior Lenticonus

Dr. Warren S. Reese exhibited a case of posterior lenticonus. The bulging of the lens was larger than in the cases he had previously seen and it contained a large opacity. These two facts probably accounted for the greater diminution of vision in this case. The

differential diagnosis of true and false lenticonus is not difficult if one has seen these cases previously. False lenticonus is really a variety of nuclear opacity causing an irregular refraction. This is easily demonstrable by noting the distortion of the fundus as one looks through different parts of the lens. The border of a false lenticonus is much less sharp, and the opacity itself, larger. False lenticonus is always seen in older people and causes an increasing myopia. A number of cases have been incorrectly reported as true lenticonus. True lenticonus is rare, whereas the false type is not very uncommon. The former is unilateral and nonprogressive, whereas the latter is bilateral and after a period of years causes marked reduction of vision, especially for distance.

Dr. Reese also exhibited a case of blood-staining of the cornea in a child who had been struck in the eye with a strap. Attention was called to the difficulty of differentiating this condition from a lens dislocated into the anterior chamber. This differentiation is particularly difficult in those cases of blood staining that have lasted long enough to permit a certain amount of absorption so that the corneal infiltration assumes the color of a lens.

Discussion. Dr. Appleman said that 2 cases of blood staining of the cornea had recently come under his observation.

The first one came to the Wills Hospital clinic because of severe pain in the eye from secondary glaucoma developing after inflammation of the uveal tract. Inspection revealed an exudate in the anterior chamber which had the exact appearance of a dislocated lens. The cornea was clear for a space of 2 or 3 mm. within the limbus all around. The contained mass was lenticular in shape, with the color of a sclerosed lens, and of about the same size.

Inasmuch as a previous operation had been performed, it was thought that on account of some accident the lens was dislocated without deliverance, and that a subsequent inflammation had resulted in exudation and interference with filtration, causing the glaucomatous pain from which she was suffering. While the patient was willing for enucleation of the eye, it was deemed advisable to try to get the supposed lens from the anterior chamber, and, failing in that, to perform an iridectomy in the hope of giving her relief.

A keratome incision was made, the point of the instrument penetrating the mass. No lens matter was forthcoming and, with a spatula, the supposed lens was found to be a mass of exudate adherent to the cornea in front and to the iris behind. Healing was prompt and the patient had relief from pain. Before leaving the hospital the exudate seemed to be breaking up. When seen after an interval of about two weeks, the mass was seen to be disintegrating and much of it had absorbed.

Dr. Appleman felt that the puncture of the mass and the interchange

allowed through the iridectomy, might have been the cause of the disintegration of the mass.

Dr. Cowan said that the case of blood-staining of the cornea, shown here, was more marked than any he had ever seen. The granules of pigment fill every part of the corneal substance, out to the anterior surface.

In Dr. Reese's case of posterior lenticonus Dr. Cowan said that of 8 cases that he had seen the resemblance is most striking. In every case the process started in the region of the posterior surface of the fetal nucleus and with optically clear spaces seen near the posterior Y. There was in each case a stream of opacities, varying in size and degree, which extended backward toward the protuberance. The base of the conus was surrounded by a gray ring. The posterior capsule was intact in every instance, with no irregularity of the double line.

In reference to the case of blood-staining of the cornea, Dr. Appleman asked whether, in the practice of those mere experienced, the opening of the anterior chamber in these cases had in any way hastened the absorption of the exudate.

In reply, Dr. Holloway said that he had never been unfortunate enough to open an eye of this character. His experience differed from that of Dr. Reese, he had never been able to see any resemblance between this condition and a dislocated lens in the anterior chamber, despite the fact that reference is not infrequently made to it. The first case of this kind that had come under his observation, was a student at the Overbrook School, and he had seen another when he was on duty at the Wills Hospital.

About a year ago he had a case of traumatism in his ward where the anterior chamber was partially filled with blood, and one could conjure up, in this case, a possible suggestion to a partial luxation, but not to the extent of making such a diagnosis.

He said that one of the interesting things about these cases is the chronicity of the staining, as it usually requires months before the cornea becomes clear. Still further, the clearing always begins about the periphery. It is not generally realized that a subconjunctival hemorrhage will also produce blood staining of the cornea in the area adjacent to the hemorrhage, and to a degree sufficient to be readily recognized, with the loupe. Dr. Holloway said that some two years ago he called Dr. Cowan's attention to this fact, and he had advised him that he had seen it in several cases; in one instance it was noted when all evidences of the hemorrhage had disappeared.

Dr. Holloway said that he knew that Dr. Griscom would forgive him if he directed a question to Dr. Cowan, and asked him, in the classification to which he had referred, whether the so-called epithelial dystrophy of the cornea was included. While the pathology of this and the types that have been referred to in this discussion is somewhat similar, the clinical picture presented is entirely different.

Concerning Dr. Reese's posterior lenticonus case, excluding medical meetings, Dr. Holloway said that he had seen but 2 cases in the services with which he has been connected and in his private practice. One of these was a patient who came to the University Hospital some eighteen or twenty years ago, which he felt sure Dr. deSchweinitz would recall. The other

was a boy of some ten or twelve years, who was observed at his office about two years ago. He said that Dr. Cowan would probably remember this case, as he also examined the patient. He will recall that there was a typical lentiglobus with the usual opacity projecting into the small posterior concavity.

Sarcoma of the Choroid, Being a Clinico-pathological Distribution

G. E. deSchweinitz, M.D., B. F. Baer, Jr., M.D. and Perce DeLong, M.D.: From a group of 25 cases of sarcoma of the choroid investigated clinically and pathologically during the last five years, the authors selected 7 for description as well as for comment on the following points:

1. The value of the ordinary transillumination test, the transilluminator being fitted with a small bulb, was emphasized, but it was pointed out that a subretinal exudation (not growth) may completely obstruct transillumination; pupillary transillumination with the aid of the beam of a slit-lamp is highly recommended.

2. Vitreous opacities, as well as considerable disturbance of the vitreous structure, are not infrequently present in cases of retinal detachment due to tumor.

3. A retinal detachment in a hyperopic eye, without history of injury or the presence of choroiditis and devoid of a retinal rent, should always be regarded with grave suspicion. A *sudden* detachment of the retina with one or more visible rents or dialyses is probably never due to sarcoma.

4. A small, circumscribed detachment of the retina in the macular area should almost surely be regarded as an early stage of a tumor.

5. Diminished intraocular tension is quite common in sarcoma, not only of the ciliary body, but also of the choroid; in these cases it is probable that the slit-lamp will reveal considerable vitreous degeneration; in short, diminished tension does not necessarily eliminate the diagnosis of tumor. Should the tension, having been low for a considerable time, begin to rise, it may be only a few points, it is a danger signal and means that the filtration angle is being blocked by exudation, the products of iritis or of sarcoma cells.

6. Although localized scleral telangiectasia is most suggestive, it should also be remembered that a patch, especially above, in all respects resembling an ordinary circumscribed episcleritis, may overlie a choroidal or ciliary body sarcoma.

7. If after enucleation extrascleral nodules are discoverd, even though no suspicious fragments are found in the orbital contents by ordinary examination, evisceration of these orbital contents is a proper procedure.

8. Postoperative radiation naturally depends upon what is found after the eyeball is removed; certainly after evisceration of the orbital contents it should be employed.

9. The vital progrognosis in cases of sarcoma of the choroid, as was shown by statistical records abroad and elsewhere, is steadily improving where early enucleation has been possible, and especially in recent years by the help of properly applied postoperative radiation.

In closing, Dr. Baer said that in the early days, irradiation was followed by contraction of the socket, but at the present time he thought that very unusual. In the *x*-ray department of the University Hospital, the following method is employed for intraocular growth, 350 mg. hours, through 3 mm. lead filter, given for 4 doses at intervals of two months. If the growth has broken through the orbit is infiltrated with 250 mg. through 2 mm. brass filter, 1 dose, unless recurrence is observed.

In cases in which there has been breaking through, it is generally the plan to eviscerate the contents of the orbit before subjecting to irradiation, and before operating for intraocular tumor, it is wiser to get permission to do a more radical procedure if necessary.

In epibulbar sarcoma the irradiation is given without filtration, 200 mc. over two areas, one for forty-five and the other for twenty seconds respectively, and we have 3 or 4 cases in which irradiation has been successful in eradicating the growth without surgical intervention.

Discussion. Dr. Cross said that in addition to the case reported by Dr. deSchweinitz, he would like to refer to the case presented before this Section March 16, 1922. The patient, Mrs. H. D., aged sixty-five years, had been operated upon by a Philadelphia ophthalmic surgeon for glaucoma. A corneoscleral trephining was performed. Eight months later, on account of continued severe pain, the eye was enucleated and a tumor noticed around the nerve at its entrance in the globe.

The chief point of interest was that on microscopic examination of the eye there was found a metastatic sarcoma at the trephined opening.

The original tumor was a melanosarcoma of the choroid, about 3½ by 9 mm. extending outside the globe. This slide was shown Professor Fuchs, who did not recall having seen one like it.

Histological Study of the Iris Inclusion Operation in the Eye of the Rabbit

Dr. Edmund B. Spaeth read a paper on this subject in which he said that a series of rabbits were operated on and kept under observation for a maximum period of twelve months. The operation included an iridectomy for control, two rabbits in which an iritis was caused with secondary glaucoma, one of which was operated on with an iridencleisis; and twelve other rabbits operated upon with iridencleisis cum iridotomy meridional, or with iridencleisis combined with iridodialysis. The paper discussed the postoperative tensions of these rabbits, and then continued with a histological study of the iris inclusions fistulæ. As a result of a study of the slides, a series was worked out showing the manner in which a fistula develops at intervals of two weeks, one month, three months, six months, nine months and twelve months. There is no doubt that the integrity of the bleb and the fistula depend upon the atrophy of the incarceration and the lining of the walls of the fistula with pigment epithelium.

The article is complete in Archives of Ophthalmology.

Discussion. Dr. Perce DeLong said that personally he was speaking purely from a histo-pathological standpoint. The inclusion acted much the same in all instances. The iris first showed evidences of degeneration of the iris stroma. This was followed by the usual inflammatory reaction with connective tissue proliferation. With the proliferation of connective tissue, we had a spongy mass which filled the perforation, and as the processes progressed, we found there was proliferation of both endothelial and epithelial cells lining the cavity.

With the shrinking of the connective tissue a channel was formed which permitted a flow of aqueous into the superficial bleb. The development of this so-called fistula depended largely upon the action of the connective tissue and the development of the endothelial and epithelial cells.

Dr. Adler said he should like very much to know if Dr. Spaeth found after doing an iridectomy, filtration depended upon the presence of iris pigment in the scar. In view of the fact that the irido-dialysis operation seems to depend for filtration on the presence of pigment, one may suppose that an iridectomy done for glaucoma would also depend upon the presence of pigment, and not simply due to cutting out a part of the iris.

In reply, Dr. Spaeth said that the one iridectomy control showed no pigment whatever in the line of incision, no bleb, small clean cut iridectomy, and no reason whatever that the tension should go down from 3 to 5 points, but it is believed the tension may have been modified by the anesthetic.

FEBRUARY 18

Unilateral Neuroretinitis of Luetic Origin with Stellate Figure at the Macula

Dr. A. G. Fewell presented before the Section a case of neuroretinitis of luetic origin with stellate figure at the macula. The patient, a colored female, aged thirty-five years, was first seen in the eye dispensary of the University Hospital on December 15, 1931. The vision of the right eye had failed rapidly for the past two weeks. There was no history of injury or of illnesses. The vision of O.D. was 3/45; of O.S. 6/6. Externally the eyes were negative.

O.D. Fine vitreous opacities. The disk was hyperemic and the margins hazed with fine striations extending out into the retina. Near the upper temporal margin of the disk there was one small hemorrhage. The veins were dark and overfull, and both veins and arteries were somewhat tortuous. At the macula there was a perfect stellate figure. The left eye was entirely normal.

She was carefully studied for any focus of infection and any renal condition. All medical studies were negative except for a positive Wassermann. The patient has had 8 injections of neosalvarsan and 2 of bismuth.

The condition is rapidly clearing up, the neuritis and hemorrhage having entirely disappeared and the stellate figure much less pronounced. The vision of this eye is now 6/9.

This case is presented because stellate figures are rarely seen in syphilis, although common in renal disease and fairly common in cases of intracranial pressure due to brain tumor.

Discussion. Dr. Holloway: These stellate figures are always interesting to those devoted to ophthalmoscopy.

In addition to the conditions referred to by Dr. Fewell, the lesion is also noted in certain cases of anemia, and I believe Dr. deSchweinitz recorded its observation in a case of chlorosis. I have seen 3 cases where this figure developed following a double perforation of the globe by a foreign body, with the wound of exit near the margin of the macular region. This is the only instance in which I have seen it as a syphilitic manifestation. While I have observed my share of diabetic manifestations, it was only recently that I saw a patient with a well-formed stellate figure, and in this patient the rays were absent between two and four o'clock but perfect elsewhere. Despite these various conditions with which it may be associated, I regard it as rare except in cases associated with choked disk, where Paton cited an incidence of 15 per cent, and in renal disease, where, if I remember correctly, Slocum's figures were between 6 and 7 per cent.

But there is another interesting phase connected with this lesion, and that is the pathology. Probably the most general opinion in regard to this interesting manifestation is that it is dependent upon degenerative changes affecting Muller's fibers which radiate from the fovea. However, Marcus Green, in describing the pegging down of the retina at the fovea, believed that mechanical tension was a factor to be reckoned with in the production of this stellate figure. Without prolonging this discussion, others have attributed it to the radiating arrangement of the cone fibers, some assumed it dependent upon the distribution of the capillaries which penetrate to the outer layers, while others have referred to the radiating distribution of the nerve fibers associated with fibrinous clots. Many have discussed the collection of albuminous fluid and the clusters of large cells containing fatty or lipoid material seen in the outer third of the retina.

While the difficulties attending a satisfactory solution of many of these retinal conditions is appreciated, the fact remains that, if one had the available material, a painstaking review and study of this stellate figure alone would make an interesting study.

Dr. William Zentmayer: I recall having seen a macular figure in a case of luetic neuroretinitis in a negro child. It had not a typical fanshape, but the round, yellow lesions were arranged in concentric circles about the fovea.

Enucleation of the Eyeball, with Special Reference to the Use of a Tonsil Snare

Dr. Luther C. Peter, presented a new method of enucleation of the eyeball, which he has been using for a number of years. The method may be used under local or general anesthesia. The technique is as follows:

The conjunctiva is detached pericorneally; the four recti muscles are severed close to the globe, with the exception of the external rectus, which is cut so as to leave a stump of about 4 mm. To this stump is attached clamp forceps for the further manipulation of the eyeball. After the conjunctiva is thoroughly detached from the globe, a Lewis tonsil snare, threaded with No. 8 wire, is introduced from the temporal side, the loop being depressed first on the nasal side and then on the temporal side, the snare being slowly tightened until the oblique muscles, the optic nerve and the vessels are severed. If the globe is ruptured, or there is any danger of rupture from any undue pressure, it is advisable to sever the attachments of the obliques by means of curved scissors before the snare is applied.

The important phases in the use of the snare are the No. 8 wire, which crushes rather than cuts the tissues, and deliberate tightening of the snare.

The advantages of the method are, first, the bloodless field which saves time in completing the operation by any method which the operator may elect to practice; second, a clean and satisfactory pouch in which to implant a gold or glass ball or cartilage; third, a minimum of trauma to the tissues in the back part of the orbit; fourth, a considerable saving of time in operating, and fifth, as much of the nerve may be included as may be required in any given case. Although the technique is simple, it is not generally known and not generally practiced.

The late Dr. John Moore, of Philadelphia, used a similar method, and Dr. C. W. Bonner, Greensboro, N. C., referred to his modification of the Lewis tonsil snare for this purpose in the *American Journal of Ophthalmology*, September, 1931, page 959.

Other points of importance in obtaining a good rotating stump after enucleation, as stressed by Dr. Peter, are, first, the importance of a gold ball of suitable size, 18 to 20 mm.; second, cleanliness of the field of operation, including careful cultures before operation, the wearing of gloves, and care that the gold ball is not touched after sterilization until it is deposited in the pouch prepared for it; third, the muscles should not be united in front of the gold sphere, and fourth, a compress bandage should be applied for five days, and renewed for a subsequent three days to insure a minimum amount of reaction. No extrusion should follow this technique.

Discussion. Dr. Spaeth: In the practice of enucleation with a snare, there are two points worth mentioning; first, the bloodlessness of the operation, for the procedure is carried on in a perfectly dry field without the necessity of constant sponging, and hence with a much lessened danger for possible infection. The second point is the ease with which one can get a long nerve stump, for with the snare slowly coaxed back a long stump can be taken out very readily.

Dr. William Zentmayer: I am pleased that Dr. Peter has called attention to the use of the snare in the operation of enucleation of the eyeball, as it is clear from Dr. Peter's experience that in certain conditions it is of distinct value; and it seems strange that it has not been more in use, since as early as 1914 H. R. Wright pointed out its advantages in securing a bloodless field for inserting conjunctival sutures. I believe that Dr. Fisher, of Chicago, has for some years employed this method.

Dr. George Cross: While I have had no experience in using the tonsil snare in enucleation, I would like to take exception to the statement that the implantation of an 18 to 20 mm. gold sphere gave the best results in the movement and fitting of an artificial eye, though it is a big improvement on simple enucleation. Dr. Greenwood, in 1921, at the meeting of the

American Academy of Ophthalmology and Oto-laryngology, made the statement: "That any individual who was to have an eye removed should have the advantage of the best cosmetic result, and that any surgeon who does not do this is neglecting his duty to the patient." I think a better result will be obtained by the Dimitry operation, using a 13 mm. gold ball in the scleral cavity. This operation was originally advocated by Dr. Victor Ray, who used a fat implant in the scleral cavity. Artificial eye fitters find better centering of the small ball in the Dimitry operation.

Dr. Peter (in closing): I have not found any reference in the literature to enucleation by means of a tonsil snare excepting the clinical report of Dr. Bonner, which appeared last year.

In reference to Dr. Cross' claim that the Dimitry operation, or implantation of the scleral cavity, offers a better rotating stump, I believe that, while the Dimitry is a very satisfactory operation which I have practiced for a number of years, the subject of this paper was that of enucleation and the best method to obtain a good rotating stump under such conditions. There are instances in which one may choose between a Dimitry and an implantation in the capsule of Tenon, and there are other instances in which implantation in the sclera cannot be practiced. In general, however, I have preference for implantation in the capsule of Tenon.

There should be no difficulty in the fitting of a shell after a gold ball implant into the capsule of Tenon. The chief obstruction in the fitting of any shell arises from the tendon of the superior oblique, which is of no special value after an enucleation. If this tendon were removed the rotations would probably be more satisfactory.

A Case of Bilateral Metastatic Carcinoma of the Choroid

Dr. William Zentmayer: The diagnosis of the ocular condition in this case lacks pathological confirmation, but the ophthalmoscopic findings in conjunction with the case history seem sufficient upon which to base the diagnosis of bilateral metastatic carcinoma of the choroid.

S. M., female, aged sixty years, consulted me June 20, 1930, because of failing vision and night blindness, which she had first noticed early in February, 1930. There was neither ocular pain nor discomfort. Glasses had been worn for near work for about ten years.

On April 1, 1930, she consulted an ophthalmologist, who ordered for O.D. + 2.00 and for O.S. + 4.00. Within a month's time these were changed to O.D. + 3.50 and O.S. + 5.00, and somewhat later to O.D. + 5.25 and O.S. + 5.25.

At the time of her first visit to me vision with glasses last prescribed was O.D. 6/15; O.S. 6/22; 3.25 added T.O. 75 was read. Tension 19 mm. Hg.

Ophthalmoscopic examination with euphthalmine mydriasis. O.D. media clear. Disk round. Small central excavation. Beginning on the nasal side of the disk near the superior vessels, and sweeping around above the disk and macula, far out into the temporal area, where it extended into the inferior part of the fundus, was a uniformly flat, yellowish-gray infiltration of the choroid producing a solid detachment of the retina. The general level of the involved area was about + 5 D. In some places it measured + 7 D. The fundus level was + 2. All the retinal vessels, especially the arteries, were diminished in size.

O.S. Media clear. Disk vertically oval. The fundus presented much the same appearance as that of O.D., but the area involved was not so extensive. Between the disk and the macular there was a more or less circumscribed area with a mottled surface upon which were small hemorrhages with new vessel formation. These were probably due to emboli of carcinomatous cells, such as were found histologically in Archangehky's case. Along the temporal vessels were areas of yellowish-white degeneration or infiltration of the retina.

In July, 1930, vision had fallen to O.D. 6/30. There was a slight increase in the elevation of the retina.

Dr. deSchweinitz saw the case and agreed that the lesions were indicative of metastasis from the breast. He advised a series of radium treatments to the eyes. One such treatment was given by Dr. P. B. Widmann, July 26, 1930.

The patient was last seen November 24, 1930. Vision was hand movements in each eye. There was further extension of the solid detachment and an elevation of 7 D.

Death occurred from exhaustion February 22, 1931.

The general medical history was that a uterine fibroid had been removed in 1925. The left breast was removed radically January 20, 1928. The pathological report was scirrous carcinoma of the mammary gland with metastasis to the regional lymph nodes.

Three series of Roentgen therapy were given to the left chest area during the ensuing eighteen months. Roentgenogram September 20, 1929, showed metastasis involving the lower half of the left pulmonary area and practically all the spine, with metastatic areas in the left lung and in the liver.

From March 9, 1930, she received weekly injections of a serum antitoxin prepared by the Murdoch Research Laboratories and

administered by Dr. J. W. Amey, of New York City. For a time there was a gain in weight and strength and a diminished lung-field density.

In 1908 Suker and Grosvenor collected 64 cases from the literature. The proportion of females to males in these cases was 3 to 1.

Since 1908, 32 cases appear in the literature. In this series the proportion of females to males is 3 to 1, and of unilateral to bilateral 3 to 2. In a large majority the metastasis was from the mammary glands.

Discussion. Dr. George E. deSchweinitz: I have personally examined 2 undoubted cases of metastatic carcinoma of the choroid, each unilateral, 1 microscopically and 1 case (bilateral) where the diagnosis was somewhat uncertain; in 2 of these patients, both women, the choroidal lesions represented a metastasis from a carcinoma of the mammary gland.

In 1 of them the lesions followed removal of a carcinoma of the breast complicated by an attack of erysipelas. The choroidal lesions, situated in the temporal areas of the fundus, did not appear as flat growths of pale gray color, but as rather irregular, elevated masses of uneven surface which looked more like exudations than solid growths, and which yielded for a time surprisingly to the influence of radium applied after the patient had passed from my personal observation. The patient died about a year later. I recommended radiation in the case to which Dr. Zentmayer referred because of temporarily favorable results in the case just referred to.

The metastasis appears to take place through the posterior ciliary arteries, and, as the essayist has pointed out, was most frequently from a carcinoma of the mammary gland (39 times in 64 cases collected by Suker and Grosvenor; 65 times in 90 cases collected by Usher).

I believe the bilateral lesions to which I referred, and which were favorably influenced by radiation, may have been, not a carcinomatous metastasis, but irregular choroidal exudates due to the toxemia developed during the attack of erysipelas from which this patient suffered, but I am not at all sure of the accuracy of this statement.

Metastatic Sarcoma of the Choroid

W. E. Fry, M.D.: Metastatic sarcoma of the choroid is an unusual condition. In an examination of the literature 9 case reports were found. The present report concerns F. J. S., male, aged forty-nine years, who was admitted to the medical wards of the University Hospital with a history of having a pigmented mole on his left shoulder. The mole had been injured a year previously and had never healed. He had become acutely ill nine weeks previously and had died shortly after admission to the hospital, of a

generalized melanosis. Examination of the choroid revealed a number of circumscribed secondary lesions.

Since metastatic sarcoma of the choroid is such an unusual condition, the reasons for considering the present case in this group should be scrutinized. Microscopic examination of the lesion of the skin showed the growth of the cells to be irregular and infiltrating in character, as would be expected in a primary tumor, while the lesions of the choroid were well circumscribed, as would be anticipated in a secondary tumor. The cells in both instances were the same in size and staining characteristics. It would be unlikely for a metastasis to occur in an already existing pigmented nevus, which would be the supposition if the lesion in the eye were considered primary. The fact that there were several nodules in the eye, sufficiently separated to rule out a continuity of growth, adds more evidence for believing that the choroidal growths were secondary. At one point the tumor cells were seen within the lumen of a choroidal vessel. The location of the largest lesion of the choroid was such, and the size sufficient, to encroach upon the macular region. This would give enough interference with the vision of the patient for him to be conscious of his ocular difficulty, had the condition existed for a long period previous to his death.

Discussion. Dr. G. E. de Schweinitz: It is well known that Hofrat Fuchs, in his famous monograph, "Das Sarcom des Uveal Tractus," has stated that metastatic choroidal sarcoma was unknown, and that he (Fuchs) had quoted Virchow's sentence: "Those organs which exhibit a great tendency to protopathic tumor formation present a very slight inclination to metastatic deposits." Similar views were held by Professor Axenfeld, and are recorded by some of the English observers, for instance, Sir John Parsons. And yet it is true that such metastases apparently do take place, as in the case studied by the late Dr. A. V. Meigs and myself in 1894, and in the patient referred to by the essayist.

The original growth in the case studied by me and by Dr. Meigs was situated in the anterior mediastinum which was the cause of the death of the patient, an Italian, aged twenty-one years, and proved to be a round-cell sarcoma. Sarcomatous deposits were found in many organs and tissues, lungs, liver, spleen, kidneys, etc., and this extensive metastasis included the brain, both choroid coats, oculomotor and optic nerves and exterior ocular muscles.

Unfortunately, no opportunity to make an ophthalmoscopic examination was afforded, but a microscopic examination of the right choroid revealed, especially to the right of the optic nerve, collections of round, darkly stained cells, with the general character and grouping characteristic of sarcoma infiltrates. A similar state of affairs was found in the left choroid, only

more extensive; the area of infiltration began 3 mm. from the nerve entrance and was 1 mm. thick.

The deposits of sarcoma cells in each choroid, but particularly in that of the left eye, give this case an importance in the literature pertaining to sarcoma of the uveal tract. The fact that these deposits were limited to a certain area of the choroid, that they were surrounded by comparatively healthy tissue not contiguous with infiltrations elsewhere located and that the bloodvessels within them, and in their immediate vicinity contain cells of the same character, afforded evidence that they should be regarded as metastatic nodules. Moreover, these deposits were bilateral, while primary sarcoma of the choroid is always unilateral.

Luther C. Peter, M.D.: Because of the infrequency of the occurrence of metastasis of sarcoma into the choroid, I wish to record a case which occurred in the Graduate Hospital about two years ago. The primary growth was in the skin of the upper arm. Metastases were multiple, and the eye was invaded only during the last two weeks of the patient's life. During this time it was interesting to notice the rapid development of the pathology within the right eye. In this instance the eye was definitely secondary and not primary. Complete pathologic studies were made. The same type of cell was found in each lesion, including that in the choroid; and the pathologists and clinicians agreed in the diagnosis of secondary invasion of the choroid, the primary lesion being in the upper arm.

MARCH 17

Intraocular Sarcoma which Filled the Orbit Before Medical Advice Was Sought

H. Maxwell Langdon, M.D.: A man, aged forty-five years, noticed failing vision in the right eye for two years and one year of complete blindness in this eye. The eye became greatly enlarged, being three times the normal size. Replacement of the cornea by scar tissue. There was no pain until the day before coming to the hospital, when a blow on it had made it very painful.

X-ray showed the orbit filled with new tissue, but no involvement of the sinuses. The orbit was exenterated and was found to contain a solid mass in which the orbital contents were embedded and partially destroyed. Healing was uneventful. General physical examination showed nothing wrong except some enlargement of the right portion of the thyroid. Two years later he died, probably of metastasis to the liver. It was unusual to find so much involvement without pain and earlier medical attention.

Sarcoma of the Choroid

T. B. Holloway, M.D., presented a case of sarcoma of the choroid, and stated that the patient presented, a male, aged twenty-three years, had just come under observation. He gave a history of having his right eye struck by a hammer three years ago. This was not followed by any inflammatory reaction and the eye remained quiet for a year. Then the vision of this eye became impaired and has grown steadily worse. Upon seeking medical advice he was advised that he had a detachment of the retina.

Extraocularly the eye was negative except that the direct light reflex of the right eye was slightly less than the left, and the indirect light reflex of the latter possibly slightly less than that of the right. There were a few vitreous opacities. Beginning about a half disk diameter from the temporal side of the disk, there was a large round and well-circumscribed solid detachment of the retina that extended over the macular region and far toward the periphery of the eye ground. At the extreme limit outward one could note a small disk-sized choroidal erosion just below the horizontal meridian. Deep vessels over the summit of the growth were rather conspicuous and in several places small, vague areas of pigmentation were seen. The apex of the detachment could be clearly seen with a + 20 D. lens. It was thought that the conditions present showed a typical picture of a sarcoma.

The field showed a slight contraction, with practically a complete loss of the whole inferior nasal quadrant, and extending to the blind spot. Careful tests were made to see whether any difference would be shown in the field corresponding to the detachment over the growth and where it sloped off its margin. A definite change could not be interpreted.

Needless to say, enucleation was advised. The left eye was negative.

Diagnosis of Double Perforation of the Globe by Injection of Air into the Space of Tenon

Dr. E. W. Spackman described a method by which air is injected into Tenon's capsule, causing the zone to be compressed by the posterior sclera and surrounding intraorbital tissue. It is then possible by *x*-ray examination, to determine whether the foreign body has

perforated the posterior sclera or remains within the globe. (Complete paper appears in the *American Journal of Ophthalmology.*)

Discussion. Dr. Warren S. Reese: Most every one who has had much experience with the Sweet method of localization will testify to its accuracy. Any trouble arising almost invariably is due to incorrect interpretation of the localization chart. In other words, the interpreter, either ophthalmologist or roentgenologist, forgets that the localization must be transferred from mathematical to anatomical terms. I have here a localization sheet on which a foreign body appears in the globe on all these plains of the eye shown on the chart, though, as a matter of fact, this foreign body is really outside of the eyeball. If the surgeon looks at this sheet casually, he will state that the foreign body is in the eye, do a sclerotomy and fail to get the foreign body. Later, on discovering that the foreign body was not in the eye, he will say that the localization was wrong. As a matter of fact, the localization was entirely correct but was interpreted incorrectly.

Dr. Spackman told me recently of a case in which a well-known roentgenologist in this city stated that a foreign body was in the eye, but was not found when the globe was enucleated. It was then discovered that the globe was microphthalmic or at least smaller than the average.

The ingenious method that Dr. Spackman has called to our attention is an anatomical method in that it places the foreign body in respect to Tenon's space. It, therefore, is not liable to the mistakes of interpretation common to the Sweet method. In the case that Dr. Spackman referred to, a young man was standing in back of and to the right of a friend who was firing a revolver, when he suddenly felt something strike his right eye, and immediately noticed loss of vision. When examined there was a wound of the cornea and lens, the latter being cataractous. The foreign body localized in the sclera posteriorly, so that we could not tell whether it was intra- or extraocular. I introduced a needle, curved to correspond to the curve of the globe and attached to a 10 cc. syringe, into the bulbar conjunctiva of the right eye near the limbus, up and in. I slid the needle along about 5 mm. and then pierced Tenon's capsule, continuing the insertion until the needle point was pretty well posterior. Almost all of the air in the syringe was then injected into Tenon's space, at the conclusion of which there was some proptosis and a bulging had just appeared over the internal rectus. The patient was then x-rayed, as Dr. Spackman has shown, and definite proof obtained that the foreign body was outside of the globe. I learned this morning that the eye was perfectly quiet and feel confident that it will remain so.

It is unnecessary, before a body of this kind, to speak of the importance of knowing whether a foreign body is within or outside the globe. We would all consider it most reprehensible to do a sclerotomy unnecessarily. Likewise, we have all seen cases where a retained foreign body has eventually caused trouble even after the lapse of many years. I hope, therefore, that the members of the Section will make use of this method in suitable cases.

Dr. A. Cowan: The Sweet Localization Chart is based on the measurements of the schematic adult eye, 24 mm. in length. This means that the

24 mm. is measured from the anterior surface of the cornea to the retina, so that allowance of about another millimeter should be made for the sclera. I do not know just how long the eye of an infant is approximately, but it is so short that if it were not for the great strength of the dioptric system, there would be a great amount of hyperopia. An infant's eye is so much shorter than an adult's that this diagram could not possibly be used in young children to localize a foreign body.

The refractive error should also be taken into consideration, allowing about three diopters for each mm. in length. Certain allowances should always be made for certain cases when using the Sweet system, which is based on 24 mm. distance from the anterior surface of the cornea to the posterior surface of the sclera.

Dr. Adler: I wish to express my thanks to Dr. Spackman for bringing this valuable test to our attention. In both the cases where the test was of value, the *x*-ray plates showed a foreign body in the neighborhood of the posterior half of the globe, either in or just posterior to the sclera. In one case the visual acuity was 6/22. An attempt to extract the foreign body would certainly lead to further visual loss. There was no response to the magnet, and it was a puzzling question whether to leave the foreign body in place or attempt its removal. The injection of air proved the foreign body to be back of the sclera, and decided the issue. The eye was left alone, and when last seen was quiet. A traumatic cataract which had subsequently developed had reduced the vision to light perception. A light field and projection were good.

It is true that not many cases will necessitate this differential test, but in the few cases it will prove very valuable.

Dr. William Zentmayer: I would like to ask whether in any one of the 3 cases reported there was a subconjunctival hemorrhage. This is not apropos of *x*-ray localization, but the occurrence of subconjunctival hemorrhage in injury by foreign body is said to be indicative of double perforation. I have observed this at times.

Dr. Langdon: I think all have been in the disagreeable position of not knowing whether a foreign body was within or without the globe. As Dr. Reese has already said, we would all consider it most reprehensible to open the eye for a foreign body which was in the orbit, and not find it, but it is equally reprehensible sometimes not to open it.

I had an experience with a case in which Dr. Pancoast had localized the foreign body and felt positive it was in the orbit, but there was a double perforation, and attempt was made to extract it, the external rectus muscle being resected. The globe was badly damaged, and permission had been obtained to enucleate the eye if the foreign body was not found. The reason for going into the orbit was that the eye was so inflamed and seemed to be getting worse, and it was felt that it was better to remove the source of irritation, but the attempt failed. The globe was enucleated and the foreign body was found within the globe, the eyeball in this case being very much longer than the standard 24 mm.

If you have made a refraction of the patient previously, it would be possible to make the calculations suggested by Dr. Cowan.

I feel that this method is of very great advantage in studying a foreign body located posteriorly in or around the sclera, and a great deal of valuable information can be obtained if it is used properly.

Dr. Spackman (in closing): Regarding Dr. Reese's discussion, that a foreign body may be outside the globe but appear within the globe on all three localization diagrams, because in the diagram we are looking at the equatorial planes. The following is a method by which roentgenologists determine this, and I believe it would be wise for all ophthalmologists to be familiar with this method. If we find that the position of the foreign body, in the diagram of the horizontal section of the Sweet Eye Localization Chart, lies in any plane except the equatorial plane of the globe, which in this case is 12 mm. posterior to the cornea, a line is drawn through the actual plane bisecting the foreign body. The radius of the actual plane is then measured by calipers and a circle of the same radius is superimposed on the front view of the localization chart. If the foreign body lies within the circle it is within the globe. If outside the radius of this circle it is out of the globe even though it may appear within the larger circle which represents the diameter of the equatorial plane. The description of this method was published in detail by B. S. Stevenson in October, 1926. In reference to the case of which Dr. Reese has spoken and in which the foreign body had been localized within the eye but on removal of the globe it was not found, it was assumed that the eye was 24 mm. in length and if, as in this case, the eyeball was shorter the above method will be of no avail.

Dr. Cowan's point is excellent, but if, as is so often the case, the anterior chamber is filled with blood and the ophthalmoscope will not help to judge the length of the eyeball.

In reply to Dr. Zentmayer's question, one case showed subconjunctival hemorrhage.

Allergic Investigations on Twenty-three Cases of Vernal Conjunctivitis

Miss Ida Teller (by invitation): The work here presented was done in an effort to determine if vernal conjunctivitis had an allergic etiology or not. There were primarily four points studied on which this decision was to be made: (1) The allergic history of the patient, that is, both the periodicity or return of symptoms at a given time of year, and the presence or absence of other allergic conditions; (2) the family history of allergy; (3) cutaneous tests to determine if the patients were sensitive to any foreign proteins, with a study of the relationship of any positive reactions to the occurrence of vernal conjunctivitis; and (4) the eosinophile count in the blood and eye smear studied for eosinophiles. A physical examination was also made on a number of these patients with special reference to any endocrine dysfunction.

The results of this study showed that a normal number of patients gave a history of other allergic conditions, although an unusually large number, or 21 per cent, gave a family history of allergy.

The blood-count for eosinophiles on 35 per cent were above normal. An unusually large number, or 11 patients out of 20, had eye smears containing eosinophiles.

Of the 23 patients studied, 4, or 17 per cent, gave positive reactions to skin tests done by the scratch method; although in only 2 of these cases does it seem likely that the sensitivity as indicated by the skin test could be the cause of the patients vernal conjunctivitis.

Observations on Eighty-seven Cases of Vernal Conjunctivitis at the Wills Hospital

Dr. Louis Lehrfeld (by invitation): Mention is made of the possible theories of the etiology of vernal conjunctivitis and clinical evidence is presented from 87 cases of vernal conjunctivitis, pointing out its similarity to other allergies caused by pollens, animal dusts and foods.

The analysis of cases indicates that 72 per cent were among males and 28 per cent among females, that 53 per cent of the cases were lid types and 47 per cent limbic; that 39 per cent were in the age group under ten, 22 per cent, eleven to twenty years; 12 per cent, twenty-one to thirty years; 6 per cent, thirty-one to forty years; and the remaining 3 cases, forty-one to fifty-nine.

Conjunctival smears from 40 cases revealed eosinophiles, the largest count being 49 per cent in a lid case.

Of 72 cases, 31 showed eosinophilia over 4 per cent in the blood, the highest count in a limbic case being 21 per cent.

A description of the disease hints that the limbic type and the lid type may be two separate diseases of allergic origin. There is pointed out that the Negro presents only the limbic type; furthermore, that the limbic vernal is limited to the spring and summer months, while the lid type may persist in a milder form throughout the winter; that vernal conjunctivitis is a self-limiting disease; and that the cobblestone description and the milky film, commonly described as characteristic, are seen only in the advanced types of the disease.

The study also describes the discharge in the advanced lid type as being typical and diagnostic of vernal conjunctivitis. It also

points out that itching is a cardinal and diagnostic feature in all forms of vernal conjunctivitis.

Radium does not cure vernal conjunctivitis, but is valuable in alleviating the symptoms and reducing the pathology.

Calcium gluconate, given internally, has a limited value in alleviating the itching. An excellent remedy proposed for the symptomatic relief is described as the "seven times treatment," consisting in bathing the eyes with an eyecup seven times a day, using boric-acid solution, and the wearing of glasses intended as goggles to shield the eyes from exciting dusts.

The survey also points out the inadequacy of the scratch method in determining positive reactions in vernal conjunctivitis, and favors the intradermal method which gives a fair percentage of positives, indicating definitely that vernal conjunctivitis is an ocular manifestation of an allergy.

Discussion. B. P. Widmann, M.D.: Whatever may be the cause of this so-called entity of vernal conjunctivitis, there are voluminous reports in literature that seem to indicate that very gratifying benefits have been obtained with the use of radium. Optimism over these results runs parallel with the good effects of radium for trachoma and tuberculous conjunctivitis.

Some observers have been so enthusiastic about the radium treatment of vernal conjunctivitis that they hold this procedure as almost specific, particularly for the acute and subacute phases of this condition.

There was evidence to indicate that many of the cases of vernal conjunctivitis showing good results were subject to season recurrences. It was thought by Dr. Lehrfeld that this apparent tendency of only transient effects justified an intensive investigation of a group of cases treated with radium. The radiological department at the Wills Hospital prepared to study the question of dosage, eye-tissue tolerance and final end-results. Technical factors of filtration and the methods of applying radium with special reference to establishing a minimum dose, time and frequency of application were considered. Dosage factors established at the Philadelphia General Hospital on the basis of vast experimental and clinical experiences were accepted as the working basis of treatment.

Desjardens (*Am. Jour. Roent. and Radiology*, November, 1931) made an exhaustive review of the literature, experimental and clinical, demonstrating the action of roentgen rays and radium on the eye.

He found experimental evidence on animals that showed damaging effects on the tissues, as well as developmental anomalies resulting from these rays, but only on the growing eye of young animals before birth or during the first and second month of life. Three months after birth the eyes of cats and rabbits were no longer affected by doses that were tolerated by the skin.

Radiologist have found the tissues of the eye tolerant to extremely large doses of roentgen rays and radium, in actual clinical experiences, of treating malignant diseases about the eyelids and nasoaccessory sinuses.

Very large doses may cause a permanent alopecia of the lids, severe conjunctivitis, blepharitis, keratitis and cataract. Desjardens found some reports of eye injury following excessive exposure, but he believes the number of authentic cases is small and the relationship between cause and effect has not been substantiated.

The rationale of radium treatment in vernal catarrh is supported by the good effects of radiation for innumerable inflammatory conditions, *i. e.*, adenopathies, boils, carbuncles, eczema, erysipelas and a manifold variety of skin diseases. The mechanism effecting good results in these inflammatory tissues is not understood, but much credit is given to the well-known fact of a profound leukocytic infiltration resulting from radiation.

Our own experience with radium for vernal conjunctivitis at the Wills Hospital is quite in accord with phenominal regressions and almost specific effects noted by others. Itching, and distress that these patients complained of, diminished rapidly to complete disappearance of symptoms. Granulation tissues resolved; the conjunctiva frequently remained injected for a period of several weeks in the cases showing marked pathology; return of conjunctiva to apparent normalcy was rapid in the cases of slight local involvement. Intensity of symptoms and local pathology were not always proportional.

Twenty cases received radium treatment at the Wills Hospital. The majority of these cases showed clinical regressions of the local pathology. Symptomatic comfort to complete relief was common. Practically all of the recurrent cases showed seasonal recurrence of symptoms, equal to the distress at start of treatment. It is significant to note that many of the cases showed no detectable evidence of local pathology; no vestige of the original character of granulation tissue, but presenting symptoms of itchiness and pain, comparable if not worse than the symptoms at the onset of treatment. Only about 50 per cent of the recurrent cases, presenting no detectable local pathology, were relieved symptomatically with further radium treatment.

These observations justify the conclusion that, while the immediate benefits (*a*) symptomatic relief and (*b*) pathological tissue regressions, may be so spontaneous as to suggest a specific factor, symptomatic recurrences should be anticipated in 75 to 100 per cent of the cases (without detectable pathological changes) and subsequent responses to radiation are extremely sluggish. This experience is not unlike the phenomenal effects of radiation in certain types of eczema which subsequently resist radiation when there is an underlying constitutional or occupational cause.

It is potentially hazardous to continue radium treatment for successive recurrences, particularly because of the risk of obtaining cumulative effects of radium. These cumulative effects occur as a result of a diffuse endarteritis of the fine capillary bloodvessels terminating in a degree of fibrosis and hyalinization that interferes with circulation. Late degenerative changes as a result of such tissue alterations may occur within two years and even later. For this reason only moderate radium treatments were given. Filtration equivalent to 2 mm. of brass, and a plaque was made so that it would cover the entire surface of the eyelid. The treatments were given in

quantities estimated as 25 per cent of a full dose and given at weekly intervals for four treatments. A small group of cases was given treatment over a period of six weeks. Recurrent lesions were treated in a similar manner. A small group of cases was treated by inverting the eyelid and the radium plaques immediately in contact with the conjunctiva. While the immediate effects of radiation appeared to be satisfactory, the uniform tendency for recurrence of symptoms indicates that there must be a constitutional factor probably allergic in character.

Dr. Alexander Clarke: There is a superficial and tantalizing similarity between hayfever and vernal conjunctivitis. Hayfever is a form of allergy in which treatment is most satisfactory and if it can be proven that vernal conjunctivitis is also allergic in origin, it might be possible to develop a similar simple and satisfactory treatment.

Allergy is the study of the unusual reaction in contradistinction to the usual reaction. For example, the usual reaction to large doses of quinine is ringing in the ears and deafness. Such a reaction can be produced in anyone. Urticaria is an unusual reaction to quinine and occurs only in sensitive individuals. In order to prove that vernal conjunctivitis is an allergic or unusual reaction to some harmless substance, such as pollen, it is necessary to discover and prove such substances beyond a question of a doubt. At present this cannot be done.

The evidence in favor of vernal conjunctivitis being an allergic phenomenon lies in the similarity to hayfever. Both occur in summer and both have manifestations in the eyes. Both have positive skin reactions to pollens, and in both other manifestations of allergy, such as asthma and eczema, are not infrequently seen. Eosinophils are present in the typical secretion from each. The evidence against vernal conjunctivitis being an allergy consists, first, in the absence of family history of vernal conjunctivitis. Dr. Lehrfeld tells me that he has never seen 2 cases of vernal conjunctivitis in the same family. While both have positive skin reactions, it is necessary to use a solution one hundred to a thousand times stronger to get a skin reaction in vernal conjunctivitis than is necessary in hayfever. In hayfever the severity of the symptoms varies directly with the amount of pollen inhaled. Vernal symptoms, on the other hand, do not follow the pollinating periods, but are influenced more by temperature. A very small number of persons suffering from vernal conjunctivitis have been given hayfever treatments, using much larger doses. The results seem to be worth continuing.

The pathology of hayfever is an urticarial swelling which can be entirely cleared up in a few minutes by the use of adrenalin, whereas the pathology of vernal, is a persistent granulation. In this respect vernal seems to be more nearly related to eczema, an allergy characterized by a persistent inflammation. Our allergy methods have helped little, if at all, in the treatment of eczema.

Such are the arguments in favor of and against considering vernal conjunctivitis as an allergy. They are not sufficient to make an unqualified decision. My own opinion is that vernal conjunctivitis is an allergy of the eczematous type, rather than of the urticaria or hayfever group.

Dr. G. E. deSchweinitz: In regard to my experience with radium in the treatment of vernal conjunctivitis, I am satisfied with the therapeutic value of this agent, especially with those varieties of this conjunctival disease with large ("cobble-stone") lesions, and I agree with the other observers, that the efficacy of radium has been enhanced if, prior to its use, the surface of the grossly developed granulation were shaved off.

Complications may arise—for instance, loss of the eyelashes and cataract —although corneal involvement is uncommon, it does occur, and may be prevented by radium.

Some years ago Dr. Shumway published a small series of cases, some from his own clinic, with excellent results, and in no case, so far as he knew, had cataract developed.

I am much interested in Dr. Clarke's remarks on radium treatment, and think it most important, as Dr. Clarke has pointed out, that radium therapy, although useful, does not prevent recurrences, and repeated use of this agent may well produce the complications to which reference has been made. I have seen one patient in consultation where cararact had formed after radium treatment, but the type of this cataract was not that which is characteristic of the radiational variety, which I have discussed at some length in a previous meeting of the Section, especially after destruction of epibulbar sarcomas.

I have often used adrenalin in a boric-acid solution to relieve the intense irritation and itching, a lotion commonly employed in this affection. Change of the patient from one climate to another was sometimes followed by astonishingly good results, very much as in the case with hayfever patients.

Dr. Shumway: I do not think that radium has anything to do with the causing of ulceration or opacities of the cornea. The only case I have ever seen with ulceration, I feel was due to mechanical irritation, from the rubbing of the growths on the lids over the cornea.

I have recently had under treatment a patient at the Lankenau Hospital with a very marked vernal conjunctivitis of the palpebral type in which there seemed to be no result after a number of treatments with radium (fifteen or more in the last year) and no reduction of the "cobblestone" masses, although the intense itching has been lessened.

Replying to Dr. deSchweinitz' question as to the cases he had previously reported, these were entirely cured by radium given under the direction of Dr. Pancoast at the University Hospital.

Dr. T. B. Holloway: Owing to the lateness of the hour there is no time to go into a real discussion of the question, but since several remedies have been mentioned I think it would be a mistake not to allude to the use of fibrolysin, as suggested by Luedde. I have found it the most satisfactory of any of the drugs that have been suggested for this condition.

Dr. Lehrfeld (in closing): In my attempt to shorten my paper I failed to mention the use of adrenalin in combination with holocain (4 grains of holocain and 30 minims of adrenalin to an ounce of water). This is used three times a day, particularly in the evening at bedtime, when the itching seems to become intense, and when patients derive the greatest comfort from its use.

Regarding the shaving or removing the granules of the lid, I have also mentioned this in my paper. In 1 case the granules were removed from one eye and the fellow eye was treated by radium. The results, which were excellent, were similar in both eyes.

I am very grateful to all who have discussed my paper and to the members of the staff of the Wills Hospital who have made this piece of research work possible.

APRIL 21

Dr. Burton Chance exhibited the following cases:

A rather large dermoid cyst containing rolls of densely black hair which he had dissected from the orbit of a young woman of seventeen. The tumor had become increasingly prominent in the past year, and a bluishness of the eyelid quite distinct. The operation was begun through the eyelid; the cyst walls being intimately enmeshed in the tissues required a very tedious dissection. Several quite stiff hairs were easily seen in the surrounding tissues having penetrated the capsule. The cyst was distended by a thick oleaginous fluid.

Also exhibited three lenses which he had extracted entire in their capsules, each showing traumatic cataract caused by penetration and lodgment of a foreign body.

And, a case of bilateral new growth at the limbus, found in an old Italian woman who had been under observation for about twenty years, because of congenital plaques in her crystalline lenses. She had not reported in the past four years. At no time in the previous history were there any signs of such vascular masses as are now present. The patient came simply with the hope that her sight might be increased by glasses and was oblivious of the presence of the new growths, which were covered by the upper eyelids. Sufficient time has not elapsed for histological study, but they are offered as instances of bilateral sarcoma or angioma of the conjunctiva.

Wound of the Globe by an Explosion of Shatter-proof Wind Shield (Presentation of Case)

Dr. H. Maxwell Langdon: Dr. Langdon presented a young man who, while driving a Ford car, enclosed with shatter-proof glass,

stopped on the roadside because of the sleet which had frozen on the windshield. To melt this he placed a candle on the ledge inside the glass to warm it and so rid it of the ice. Shortly after this there was an explosion, and a small hole about 2 inches in diameter appeared on the inner plate and one about one-third this size in the outer plate, while at the same time the driver felt a blow on his right eye, which began to bleed. It was found that he was in need of hospital attention and was sent to the Presbyterian Hospital. Examination showed that there was a small cut at the center of the upper lid near the margin, and that the pupil was irregular in outline due to a penetrating wound of the sclera near the upper limbus. The wound was in median line about 3 mm. long, extending upward from the corneo-scleral junction, with a small bead of iris protruding. There had been a small hemorrhage into the vitreous which had rendered it somewhat cloudy but did not prevent a view of the fundus, which showed no other injury. The protruding iris was excised and the wound healed promptly with perfectly normal vision.

It is not an unusual thing for some artificial heat to be applied to sleety windshield and this, of course, may be done with impunity where ordinary glass has been used. The accident which happened to this patient shows that where shatter-proof glass is used greater care must be exercised. The material which is between the two layers of glass is a cellalose product which, when heated, generates a gas of great expansive force and it is this which produces the explosion. As this knowledge does not seem to be a common thing, it seemed well to put this case on record as a warning of possible danger.

Melanotic Sarcoma of the Choroid Occurring at the Optic Disk in an Eye with a Previous Detachment of the Retina

H. Maxwell Langdon, M.D., presented the case of a man first seen in October, 1929, with a history that the vision of the right eye had been blurred for two years. Three months previous to being seen by the writer, he had been told at the Wills Hospital that he had a detachment of the retina. Examination showed that there were no exterior changes in either eye. The vision of the right eye was 1/60 and of the left 5/4. With the ophthalmoscope the right eye had clear media, a partially atrophic disk with a fundus

level of −1, with a detachment of the retina over the whole lower portion. The highest point being about −10, with nothing to suggest any intraocular growth. Transillumination over the detached area gave a good light on all parts. He was seen again in December, 1929, with the condition unchanged, including the field except for a slight loss on the temporal side. He was not seen again until February, 1932, with a history that he had had an intense pain in the damaged eye for the preceding two days, the eye being totally blind. Examination showed that there was considerable injection of the conjunctiva of the damaged eye; the pupil was 5 mm. in diameter with no response to external stimuli, the media being too hazy to permit a view of the fundus. The vision of the left eye was 5/6 partly; pupil sluggish but responding to light and accommodation. The media clear, disk pale and almost completely cupped about 1 diopter, the fundus otherwise being negative. The tension of the right eye 47, the left eye 36; he was placed on myotics and advised to have the right eye enucleated. Two days later this was done. Healing uneventful. The tension of the left eye was reduced to 26. The vision improved to 5/5 plus.

Pathological reports show a growth at the optic disk; the microscopic report being melanotic sarcoma. No new tissue being shown in any other portion of the globe except in this one compact mass, which could not possibly have been present in the early stages of the retinal detachment without showing itself to the ophthalmoscopic examination. It is, of course, not unusual to find in a case of retinal detachment with secondary rise in tension that an interocular growth has been the starting point of the whole proceeding; but here we have an eye with what seems to have been a simple detachment of the retina with a neoplasm developing in the eye later in the course of the condition. It is the rarity of this sequence of events which seems to justify the recording of this case.

Microphthalmos with Coloboma of Iris, Choroid and Disk

Hunter W. Scarlett, M.D.: Miss A. W., aged thirty-one years, presents a microphthalmic eye on the right side with a coloboma of the iris, choroid and disk, and also a small congenital cataract. The left eye shows a coloboma of the iris and choroid. Vision O.D. Hand movements. O.S. 6/30 cc 6/10.

The fetal ocular cleft normally closes early in fetal life. If there

is a defective closure in the extreme posterior part of the cleft there is a coloboma of the optic nerve sheath; if a little further forward, a coloboma of the choroid; or if near the anterior lip of the cup, a coloboma of the ciliary body and the iris results; thus analyzed, it is evident my patient shows a defect along the entire length of the cleft, as she presents a coloboma of all the structures mentioned.

Concerning Leber's Disease

T. B. Holloway, M.D., presented a paper entitled "Concerning Leber's Disease"—will appear in full in *Archives of Ophthalmology.*

Discussion. Dr. William Zentmayer: Soon after Mr. Fisher advanced his theory as to the possible etiology of Leber's disease, I reported a history of two families in which two male members of each were affected.

All four were radiographed by Dr. Pancoast and all showed either a high normal or an increase in the size of the sella. As a control, Dr. Pancoast radiographed the pituitary region of the sister of one of the patients and on finding that the sella was enlarged, the eyes were examined and she was found to have a small relative central scotoma.

As in Dr. Holloway's one case, in one of my cases the scotoma had broken through above, and it is probable that a lesion of the pituitary would have been considered had it not been for the family history.

I do not see why Rehsteiner should consider the degeneration that he found in the papillomacular bundle no proof that it is due to a toxin from some endocrine disturbance as the findings were exactly the same as found in certain forms of toxic amblyopia.

It seems an unusual coincidence that only this week I received from Mr. Fisher a letter in which he has the following to say about the theory he advanced in regard to Leber's disease.

"As regards my view of the pathology of Leber's atrophy, I have nothing new. I have always regretted that those who have considered my proffered explanation have tended to ignore the arguments upon which it is based, and concentrated on the one point whether radiography showed or did not show any change in the sella turcica; positive changes shown by *x*-ray would be very confirmatory, but their absence by no means disapproves my view; indeed, any gross bony change is not to be expected."

It is difficult to reconcile recovery of function in a condition considered to be an abiatrophy, as by the term abiatrophy is meant a condition in which the cells at birth have vitality enough to exist only until they have their full development, and at that time begin to undergo degeneration.

As pigmentary degeneration of the retina occurs more frequently in the male than in the female, it has been suggested by Wibaut that the administration of a female hormone might supply a deficiency in those males affected, and good results have been claimed from its use. As Leber's disease shows the same sex predominance a similar therapeutic suggestion can be made.

Dr. H. Maxwell Langdon: Two men for whom I have the utmost respect have put forth an explanation for the occurrence of Leber's disease and ascribed it to abiotrophy. Of course, I refer to Sir William Gowers and Mr. Treacher Collins.

Abiotrophy, or congenital deficiency of the vitality of the tissues, signifies a degenerative process from the very first, with steady development of the atrophy until the condition terminates in complete loss of function. One of the best types of this thing in ophthalmology is, I think, pigmented degeneration of the retina, where we have a degenerative, noninflammatory, steadily progressive process from the first.

If Leber's disease is abiotrophy, in other words a degenerative process, I cannot understand how we can have a case like that which Dr. Holloway has just described; in other words, how a case can progress steadily towards visual loss as this did, and then make such a remarkable improvement, regenerate and have function restored.

It seems to me that abiotrophy will not explain the conditions which have existed in his case, and we must look elsewhere for a satisfactory reason for the course of events.

To me, Dr. Holloway's case must have been on an inflammatory nature to have had the course which it did.

Rehsteiner believes that his case is the type which was atrophic from the first, without signs of inflammatory involvement at any time during its course. If he is correct, his report, taken in conjunction with the clinical history of Dr. Holloway's case, would make it seem that there are two different processes with which we are dealing, and that one explanation will not cover the whole field.

Dr. Burton Chance: I have seen five members of the family from which Dr. Shannon, tonight, has presented two sons exhibiting signs of degenerative disease of their optic nerves. I found three affected, but could not elicit any information concerning the probability of there being other cases in the family.

I saw the first in 1929, and visual acuteness in the right eye was 4/45. and the left 4/30, improved by glasses, in spite of advanced atrophy in each eye. Radiographic studies showed thickening of the foramenal regions which were regarded as hyperplastic in character. This case tonight I first saw when a boy of thirteen, and I did not see him again until tonight. The next member was examined in 1931 whose visual acuteness was 4/4, with only narrow absorption lines at the temporal borders; his condition is practically unchanged. The middle boy, aged seventeen, had 4/4 in his right eye, and amblyopia in the left. No lesion was demonstrable by ophthalmoscope. There was, however, a central scotoma in the right eye; and, at the end of two months, the disk was not quite as clear as at the earlier examination, and the visual acuity was 1/60. He was last seen June 30, 1931.

The examination of the mother disclosed no such symptoms. The father had met with an accident, hurting his foot in falling from a ladder. His vision was 4/12 right, 4/20 left, improved by glasses to 4/5 and 4/6. Nothing in the eye grounds of special interest. He was quite neurotic, maintaining

that his fall had made it impossible for him to undertake any work and that he could not see, although the glasses enabled him to see at the standard. His nerve-heads, when seen tonight, are as healthy as they seemed to be when observed last in June, 1930.

I find it difficult to place these cases among the hereditary atrophies, as there is not a clear connection with a previous generation. Certainly they are essentially atrophic, which atrophy I consider as being dependent upon some peculiarity of nutrition in each of those affected, rather than as an example of the effects of an inherited tendency.

I should like to ask Dr. Posey if he recalls the history of the occurrence in a family which he reported several years ago?

Dr. William C. Posey: I saw three generations of a family with this disease. The primary case was a young man aged about twenty years; also his uncle and his grand-uncle, all three had Leber's disease.

In searching for a cause of the affection I looked into the literature and was impressed by a theory advanced by Berger, of Paris, that Leber's disease was probably caused by an anomalous condition of the sphenoid bone, the optic nerve being affected in its passage through a too narrow optic foramen. All 3 cases I saw had very flattened occiputs and it seemed as though the condition might have been due to anomalies in the sphenoids.

I shall ask Dr. Holloway whether this theory of the origin of Leber's disease has been entirely given up. Now that a New York colleague has examined the optic foramina by x-ray it would seem that we would now be in a position to determine something definite about any anomalies in the size of these passages.

Dr. Shannon: The diagnosis of Leber's disease in these 2 cases may seem doubtful, as no evidence of this disease could be traced in the parents, the grandparents, nor the greatgrandparents. On the other hand, may we not conceive it possible that this condition may have originated in this generation?

Furthermore, the history of the cases, appearance of the disks, the typical fields, together with the physical and laboratory tests, would seem to point indisputably to hereditary optic nerve atrophy.

Therapy in Leber's disease is, of course, of doubtful value. The suggestion of diathermy was made by Dr. Schmidt, who referred the cases to me, and I saw no reason why it should not be tried. In addition, anterior pituitary products, high frequency and strychnia have been employed. The improvement in vision is encouraging, although it may be merely a remission which sometimes occurs in these cases.

Syphilis in the Third Generation

Joseph V. Klauder, M.D. Syphilis in the third generation is very rare, but few cases have been reported. Two children from the Wills Hospital Clinic for the treatment of ocular syphilis were presented. One child had interstitial keratitis, which was probably third generation syphilis. The other child did not have interstitial

keratitis, but had syphilis which was regarded as third generation syphilis. The case records are as follows:

Girl, aged twelve years, oldest of four children. When first seen had typical interstitial keratitis of both eyes. The second eye became involved one month after the first eye. Clinical examination was essentially negative excepting for overweight and infected tonsils. There was no stigmata of congenital syphilis. The Wassermann reaction was 4 plus, the luetin test was positive. Two younger brothers and one younger sister were negative on clinical examination and the Wassermann reaction of each was negative. Her father denied ever having had venereal disease, clinical examination was essentially negative. Wassermann reaction, 4 plus. The Wassermann reaction of his wife was negative; there were no miscarriages. The father had seven brothers and sisters, none dead; one had a hemiplegia at the age of thirty-one. His mother died of cause unknown to him. His father (patient's grandfather) had tabes and is now dead. The physician who attended him informed me as follows: "I examined Mr. W., five years ago. He presented very definite signs and symptoms of tabes dorsalis, typical tabetic gait, loss of bladder control, Argyll-Robertson pupils, absent patella reflexes, in fact, the disease was quite advanced. The blood Wassermann and spinal fluid were not examined. The blood Wassermann of his son (patient's father) was 4 plus. Suggested examination of his brothers and sisters refused."

Case II. Girl, aged nineteen years, only child. When first seen had typical active interstitial keratitis. She presented none of the stigmata of congenital syphilis. Her Wassermann reaction was 4 plus. She is married, has had one miscarriage and one child aged sixteen months. This child is well nourished, clinical examination essentially negative. The blood Wassermann reaction is 4 plus; the luetin test is positive. The patient's mother (baby's grandmother) had one miscarriage, no still-births. She gives a definite history of having been infected with syphilis soon after marriage, of having had a secondary eruption, of positive Wassermann reactions and of having had antisyphilitic treatment. Her blood Wassermann now is 4 plus. Her husband died of cancer. The hospital where he died informed me that the Wassermann reaction was negative.

The first case, although the child's grandfather had tabes, acquired syphilis of the father cannot be definitely excluded, hence third-generation syphilis in the child is not certain. In the second case, however, third-generation syphilis is more likely, since the child's mother had interstitial keratitis and the grandmother gave a definite history of infection and her Wassermann was 4 plus.

OCTOBER 20

Dr. Mary Buchanan brought before the Section twin girls, aged nine years, who had come to the clinic for refraction, and on exami-

nation both of them showed retained pupillary membranes. In one of them it is seen at 8 o'clock, and in the other between 10 and 11; in this one there are two branches, one of which is connected with the capsule. The vision is 6/9, corrected to 6/6. Both were three weeks' premature. Nothing is wrong with the fundus.

Dr. L. F. Appleman brought before the Section a case of pulsating exophthalmia which came under observation at the hospital, on October 5, stating that ten days previously had been struck with a fist on the right eye.

The following morning had soreness over right side, and headache over temporal region. In three days noticed diplopia with complete oculomotor and sixth-nerve paralysis. Ptosis was complete and has continued. There is a distinct bruit heard over the temporal region and also upon palpation over the globe. Ophthalmic examination showed the veins very tortuous, but no hemorrhage. There is no optic neuritis. The spinal-fluid examination showed abundant red corpuscles. Examination of smear from the eye itself showed staphylococcus albus and streptococcus desidens. Spinal reaction negative to Wassermann test.

X-ray revealed no fracture; two plates were taken, but in neither could fracture be detected.

He has been kept under observation, given potassium iodide and rest. Probably, as in many of these cases, the solution of the problem will be in ligation of one or more branches of the carotid artery.

Discussion. Dr. William Zentmayer: What effect has pressure on the carotid artery?

Dr. F. H. Adler: I should like to ask for an opinion regarding the cause of the proptosis in pulsating exophthalmus. The usual explanation given it that it is due to venous engorgement, but the retinal veins in this case are only slightly dilated, nor are the episcleral veins or the veins of the lid dilated. It does not seem possible, therefore, that venous engorgement of itself can account for the protrusion of the globe.

Dr. Appleman: In answer to Dr. Zentmayer's question as to what effect the pressure had on the carotid; this has not been determined.

Amaurosis After Uterine Hemorrhage with Restoration of Vision Following Transfusion

Dr. H. Maxwell Langdon and Dr. George M. Laws: A patient, aged thirty-nine years, was admitted to the Presbyterian Hospital, March 14, 1932, with the history that for two years there had been

spells of recurrent vaginal hemorrhages lasting from three to fourteen days, the last attack started fifteen days before admission and at times had been very profuse.

Vaginal examination revealed a large, soft mass attached to the left side of the canal.

Hemaglobin, 45 per cent; red blood cells, 1,800,000, and white blood cells, 22,000.

The day after admission she was given 300 cc. of citrated blood intravenously, with improvement in her general condition. The third day after admission the patient became drowsy and somewhat delirious. Another transfusion of 500 cc. of citrated blood and 500 cc. of 20 per cent glucose was given, with some slight improvement. On the fourth day another transfusion of 500 cc. of citrated blood was given.

The patient complained of blurred vision and an ocular examination was made. The pupils were sluggish to light; the media were clear, the disks were covered with a white, fluffy material, the margins being completely obscured; there were no intraocular hemorrhages. The following day her general condition had improved, the blood count having risen to 2,719,000 and she could count fingers at 2 feet. Her general condition continued to improve; but on the ninth day vision was reduced to questionable light perception and no pupillary response to light. The fundi were generally pale and the disks less edematous. Two weeks after admission the vision was one-third of normal and the disks much less edematous and a little better color. The pupils responded well to light and there were no ocular palsies.

A hysterectomy and appendectomy were performed. The patient made an uneventful recovery, and four weeks later the vision was O.D. 6/12; O.S. 6/6. The form fields were constricted to 40 degrees. Four months after discharge from the hospital the vision was O.D. 6/7½ O.S. 6/6. The disks were slightly pale, but otherwise the fundi were negative. The patient's main complaint is "that things are dark," and with the disturbance of the retinal cell elements which, Ward Holden has shown, occurs in this type of case it seems very probable that there has been a disturbance of her light sense. Unfortunately, the patient's mental condition makes these tests very unsatisfactory.

Conditions similar to those in this case are a well-recognized medical entity and usually end in serious loss of vision if not blindness.

The loss of vision following anemia from loss of blood seldom if ever occurs after a traumatic loss of blood, but almost invariably in diseased conditions. It may occur after prolonged hemorrhage, as in this case, or after a sudden much larger loss of blood. It has been reported after hemorrhage from the lung, from the gastro-intestinals as well as the genitourinary tracts. Before transfusions became a standard treatment for such acute anemias the prognosis for vision in these cases was very bad, as the affected retinal and cerebral cells went on to degeneration, with ascending atrophy of the optic nerve before the blood condition could be brought to a stage which furnished them sufficient nutriment; the treatment, of course, being much more prolonged and slower in its results. It is not likely that ophthalmologists would be the first to see such a case, but it should be borne in mind by any physician, irrespective of his special interest in medicine, that massive transfusions offer a method of preservation of vision which cannot be duplicated in any other way and should be resorted to at once. The difference of the vision in the patient here recorded, after large transfusions were used, was so prompt and extreme that it would seem that a good prognosis could be given in any case if transfusions are made use of promptly. It would also seem that the anemia is the sole factor in the production of the neuroretinal condition, and where this is overcome no other factor remains to be considered.

Dr. Laws: There is very little to be added to Dr. Langdon's report that would be of interest to the Section. We have rarely had patients whose hemoglobin fell to 27 per cent, but I do not recall any other profoundly anemic patient who showed similar visual or mental disturbance. The fibroid tumor was unusual in that it was situated wholly within the cervix. The bleeding was controlled by packing repeatedly. After five transfusions the hemoglobin reached 55 per cent, with the red corpuscles in proportion.

Discussion. Dr. William Zentmayer: I have seen 2 of these cases. In both instances there was some underlying cause which had reduced the vitality of the ganglion, with a resulting amaurosis. In 1 of these cases the patient had chronic interstitial nephritis and the amaurosis supervened upon metorrhagia. In another case, in a man who had a Wassermann of 4 plus, the hemorrhage resulted from a duodenal ulcer. In both of these cases I feel that the systemic condition was a contributing cause of the amaurosis. In traumatic cases, even with severe hemorrhage, amaurosis is rarely seen.

Dr. Shumway: I should like to ask Drs. Langdon and Laws two questions. First, whether the anemia present showed any characteristics indi-

cating that it was a primary anemia, and not a secondary one, and whether there was any evidence of distortion of the red cells. Second, what was the condition of the appendix when removed? Was it diseased, or was it removed simply as a routine measure at the time of the operation?

I agree with Dr. Zentmayer that in most of these cases of loss of vision that are caused by hemorrhage, and are followed later by optic nerve atrophy, there is some other underlying factor, and I wonder whether in this case the focus of infection might have been in the appendix, with a resulting retrobulbar neuritis which could not be detected on account of the mental condition of the patient?

Dr. G. E. deSchweinitz: I should like to refer to a patient whose clinical history I reported to the Section some time ago. A criminal abortion had been performed, followed by severe hemorrhage which continued for six days, when, preceded by a violent headache, blindness developed (only light perception).

The ophthalmoscope revealed very pallid, double choked-disks (plus 4D.) similar to those which Dr. Langdon found in his patient. The disk-edema subsided and at the expiration of three weeks vision, R. E. was shadows, L.E. 4/15, and a visual-field test revealed an impure nasal hemianopsia—a type of field defect which has been observed in cases of this character, indeed also in cases of subsiding papilledema caused by increased intracranial pressure.

I did not again see this patient, but Dr. Maxwell Hermann, who had referred her to the University Hospital, later had the opportunity of examining her and found a satisfactory restoration of vision.

I wish to emphasize the fact, referred to by the essayist, that blindness very rarely occurs after a pure traumatic hemorrhage, and hence the belief that bleeding *per se* is not the sole etiological factor. An yet in experimental work on normal animals (Holden's research, for instance) blindness has followed excessive bleeding, which would seem to indicate that it may be the only causative agent.

Dr. Langdon's contribution to the therapeutic management of his patient represents an interesting and important observation.

Dr. Maxwell Herman: One of the things typical of that fundus picture, to which Dr. de Schweinitz refers, was the pallor around the maculæ and cherry-red spots which could be thought characteristic of embolism of the central artery. I should like to ask if macular changes of this nature were noted.

I had another case of similar nature which followed a postpartum hemorrhage in a normal obstetrical case, with the simple history of the patient complaining of the sudden loss of light. It was the typical picture, such as the case described by Dr. deSchweinitz, which was followed by optic neuritis, swelling of the nerve head, marked pallor and typical macular pallor with cherry-red spot. Dr. deSchweinitz's patient was lost track of because of a bad social history; in fact, she had registered at the hospital under an assumed name. After seventeen days she was reëxamined and it was found that she had vision enough to read ordinary print.

Dr. Langdon (in closing): My recollection is that the appendix was

practically normal. I agree with Dr. Zentmayer, and have always felt that these cases had some underlying factor which was directly connected with the hemorrhage and resulting anemia. My reason for the last statement in my paper, which contradicts that opinion, was we had such a very prompt recovery, the difference between the patient counting fingers and ability to read some of the type on the Jackson card, which is used to test in the ward, three or four days later, and not the result of any treatment except that directed toward the relieving of the anemia. Whatever the factor was, it either disappeared spontaneously without any treatment directed toward it, or was so closely associated with the anemia that recovery from the blood condition overcome the other factor. The response was so very prompt that it really makes you think it must have been the anemia without any other direct factor involved.

In reply to Dr. Herman; the appearance of the macular region showed white edema with veiling of the disk and a generalized fundus pallor, but no accentuation of the pallor over the central portion, no evidence of the cherry-red spot at any time.

Dr. Laws in reply to Dr. Shumway: Repeated blood examinations showed that the anemia was a purely secondary type. The appendix was taken out, partly as a routine measure and partly because the patient had had some vague pain in its region. The laboratory report was "chronic appendicitis," but there was no evidence that it was capable of causing toxemia.

I might add that the patient had an elevation of temperature that averaged 100° Fahrenheit during the first twenty-four hours after admission and rose to 103° after a minor operation, severe hemorrhage and intrauterine packing. This was associated with deep coma. The fever may have been a manifestation of local infection. It was a fibroid tumor of the cervix, about 8 cm. in diameter and accessible to palpation within the rim of the cervix and, therefore, readily accessible to external infection. I think, however, that we should lay stress on the fact that, while fever was present, the response to transfusions was striking, as Dr. Langdon has said, and was also striking in that she snapped out of the coma.

The first operation consisted of simply securing a specimen for biopsy and packing the cervix and vagina. The radical operation was performed several weeks later.

Dr. Burton Alexander Randall as an Ophthalmologist

Burton Chance, M.D.: In a sketch of the life and works of the late Dr. Randall, to the present generation of practitioners, he was known as an otologist, yet in his early professional days he was distinguished among ophthalmologists. His greatest contribution to ophthalmology concerned refraction and its problems. His studies in this department of the science were carried on chiefly among groups of young persons, as in schools and colleges, his first

report comprising an analysis of the refractive states of a class of medical students. Randall employed skiascopy and ophthalmometry as well as the subjective methods. The resulting analyses confirmed the dictum of observers in Europe that, clinically, myopia in the great majority of instances was a stage dependent upon the strain consecutive to uncorrected hyperopia, especially hyperopic astigmatism of low degrees. In pursuance of such findings, Randall joined others who had urged that the eyes of school children and adolescent should be systematically protected by properly adjusted spectacle lenses. Randall lived to see the world's recognition of America's distinct contribution to the science, and the fruits of his labors in time were ripened and, today, the principles which he so strongly advocated are inherent in the fabric of ophthalmology.

Randall was an expert ophthalmoscopist and he reported numerous anomalies of the ocular fundus which he depicted artistically and skilfully with pen, pencil and brush. In the meantime otologic subjects as well as the observation of the aberrations of the motility of the globes had begun to engage his attention. So also was he interested in histological technique and he reported numerous cases of disease which he studied microscopically. He was a neat and careful operator on the eye, preferring the "Holschnitt" of von Jaeger to the von Graefe cataract knife.

Randall was an effective teacher. Students and colleagues of his attest to his proficiency in demonstration in the classroom in the elucidation of the problems of physiological optics, for which he employed ingenious appliances of his own devising and construction. Both in his lectures and in the presentation of his own reports, and the discussions at scientific gatherings, he illustrated his points by his own photographs, colored sketches and drawings. Randall was of great service in consultations. He began the study of medicine at a time when it was deemed best that one should have experience in general subjects, and, as he progressed in his otological practice, he was one of the first to point out the frequency of the association of ocular diseases having a basis in nasopharyngeal and aural derangements. His helpfulness in this field readily presented itself.

Randall was a man of rare culture; his manners those of the gentlemen of the old-school. His courtesies to patients and colleagues were extended easily and widely. In time his practice became more and more confined to otology, yet as he looked back over his twenty years of devotion to ophthalmological subjects, he could rest con-

tent that he had rendered valuable service to that branch of medical science, as the perusal of the lengthy list of the titles of his papers, reports and discussions will disclose.

NOVEMBER 17

Exhibition of a Case Presenting Disjunctive Ocular Movements

Dr. Warren S. Reese presented a case of this condition. Male, aged fifty-eight years, came for refraction during the course of which it was noted that abduction and adduction of the right eye were limited, particularly the former, though on elevation this eye diverged somewhat. The patient could converge or diverge the eyes at will, most of the movement being in the left eye. During the divergence the pupils contracted and exhibited hippus. There was also an associated eye-ear movement on each side.

Discussion. Dr. Adler: This interesting case recalls a similar one reported by Dr. Spiller in 1927. The patient was a colored boy, about twenty years of age, who could hold his eyes straight and then allow either the right or the left eye to drift out or diverge, the other eye maintaining fixation. I use the word drift, as we thought at first that he accomplished this by relaxation of an internal rectus. It was soon found, however, that this was not strictly correct and that he actually turned the eye out by contracting the external rectus muscle. When either eye diverged the pupils dilated, and contracted again when the eye was brought back into the primary position.

I retinoscoped him under euphthalmine, and while one eye was divergent had him read with the fixing eye at the usual reading distance. I could note an increase in the dioptric power of the divergent eye of about two diopters, showing that when he accommodated with the fixing eye he contracted the ciliary muscle of the divergent eye about an equal amount. He was able to dissociate accommodation and convergence, therefore.

Dr. Spiller considered this case illustrated a reversion of type. You will recall that in lower mammals one finds the eyes have independent movement. They do not work together in associated movements and histologically Perlia's nucleus is not seen. As we ascend the scale, and come to those animals where the eyes are situated in the frontal plane, and can be used together, we find the first appearance of Perlia's nucleus and a very close association between the movements of the two eyes.

Perhaps it is not facetious to call attention to the marked voluntary ear movements which this man shows, which certainly is a reversion to the quadruped.

Dr. Langdon: I have two patients, one with perfectly normal vision, very low error of refraction and excellent muscle balance; retinal condition good. With the eyes fixed straight ahead, she can turn either eye in at will and not turn the other, and at the same time accommodate with either eye with the eyes in that position.

The other case can allow his eye to go outward—whether there is definite adducting power, I have not been able to determine. He can also accommodate with his eye in that position. He has a moderate error of refraction in each eye. Neither of these patients are inconvenienced by this condition, and they do not appear unless they voluntarily cause them.

A Case of Primary Sarcoma of the Iris. Removed by Iridectomy

William Zentmayer, M.D. presented a patient, aged twenty-eight years, female, who was referred by Dr. H. C. Fulton with a diagnosis of probable sarcoma in the right eye. There had always been a dark spot on the blue iris. Two months before her visit it began to grow. The eye had not been injured, inflamed or painful. There was no family history of cancer. The left eye was congenitally amblyopic. Vision equaled 6/30.

Low power biomicroscope shows a chocolate-brown mass involving the iris in its lower outer sector, irregularly rhomboidal in form with a narrow triangular extension parallel to the pupillary border. It measures 7 mm. at the angle of the chamber and 3 mm. at the pupillary border. The surface is foliated, and at the summit, midway between the angle and the pupil, is a crater-shaped depression. One rather large vessel passes to the growth from above. There is an extensive lacelike network of retained pupillary membrane. The lens shows peripheral club-shaped opacities.

Under homatropine the pupil dilates evenly. No involvement of the ciliary body can be made out. Transillumination over the ciliary body is negative. The tension is 25 Hg. 5.5 weight. (Schiotz.)

On February 17, 1932, under general anesthesia, a keratome incision was made above the upper limit of the growth large enough to admit one blade of the scissors, and the incision was continued around the limbus to 4 o'clock. A blunt Tyrrell hook was introduced, the sphincter was engaged and the entire mass with the iris was drawn well outside the anterior chamber and abscised.

Following the operation Dr. G. E. Pfahler gave 10 roentgen-ray treatments to the eye.

When the patient was last examined, on October 8, 1932, vision

with a -1.50 -0.75 c ax 70, was 6/5. There was a coloboma with clean-cut curved pillars involving about 1/5 of the iris. The visual field was full. The tension was 22 Hg.

Dr. Perce DeLong submitted the following histological report: "The specimen is composed of smoothly arranged, elongated or spindle-shaped cells and round cells, very uniform in size. They are supported in bundles or whorls by a delicate stroma with thin-walled bloodvessels. There is some melanin and iris pigment present scattered through the structure." Diagnosis, mixed-cell sarcoma.

Discussion. Dr. deSchweinitz: That a sarcoma of the iris, provided it is small and in an early stage of its development, can be successfully removed by a broad iridectomy is well known. In such circumstances this operation has received the sanction of a number of ophthalmic surgeons who write or have written with authority, for instance, Ernest Fuchs and Salzmann; and Dr. Zentmayer's interesting report again demonstrates the availability of this operative procedure.

On the other hand, other authors, for instance, Brown Pusey and Wood, are emphatic in their advice that the globe shall be enucleated as soon as the diagnosis is satisfactorily established.

I have not, personally, had the opportunity of removing an iris sarcoma by means of an iridectomy, but had in consultation with the surgeons in charge in two instances, in agreement with their recommendation, advised excision of the growth. This operation was performed, and although a number of years have elapsed, so far as I am aware, there has never been any recurrence or metastasis. There was a third case in which I recommended a similar procedure, but I am unaware of the subsequent history.

Slitlamp study of an iris tumor is of real value in reaching a determination as to whether it should be removed by an iridectomy or not, inasmuch as the iris tissue surrounding the sarcoma nodules may be intimately investigated, and any, even minute, lesions detected which otherwise might readily escape observation.

I feel that in proper circumstances, such as in Dr. Zentmayer's patient, the growth can be removed by an iridectomy and the globe not sacrificed.

Dr. Zentmayer (in closing): I believe there are cases where enucleation is the better procedure. Recently I saw a woman of fifty-eight years with a primary sarcoma of the iris. In this case I enucleated the eye because of her age and the fact that there was good vision in the other eye.

A Case of Bullous Keratitis Successfully Treated by Iridectomy

Dr. William Zentmayer presented a man, aged seventy-one years, on whom he had done an iridectomy in April, 1931, for relapsing

bullous keratitis of the right eye. The eye has remained quiet since and the epithelium in place.

In October, 1931, the left eye became similarly affected. Despite local treatments the blebs recurred and in January, 1932, an upward iridectomy was made. After a period of six weeks no further bullæ developed.

Both lenses show exfoliation of the anterior lens capsule and dystrophy of the endothelium.

Only in the right eye was the tension in the least elevated at any time.

The corneal condition was not due to glaucoma or iridocyclitis, but was probably brought about through the action of toxins on the endothelium, breaking down the barrier to the passage of aqueous through the corneal lamella, which resulted in the elevation of the epithelium.

The iridectomy was based on empiricism, the thought being that the circulation of the fluids of the eye, and consequently the metabolism of the cornea, would be improved by an iridectomy.

Discussion. Dr. Adler: This patient came to the Wills Hospital and was seen in Dr. Baer's clinic. I did not consider him a case of bullous keratitis, although this is the diagnosis on the clinic record. Since my attention was centered largely on the peculiar changes in the anterior lens capsule which one could see with the slitlamp, somewhat resembling zonular lamella. I believe that he was either a case of so-called "glaucoma-lenticulo-capsulare" or a case of endothelial dystrophy. The changes in the lens capsule plus the tendency to a slight rise in pressure led me to believe that he belonged to this type of glaucoma recently described by a number of German clinicians. At the same time the marked changes on the endothelial surface of the cornea suggested the diagnosis of endothelial dystrophy.

We did not consider doing an iridectomy as the tension was only slightly elevated. Although I was not particularly struck by the bullous character of the corneal lesion, the record plainly shows that such bullæ existed. Whatever the condition is, the iridectomy which Dr. Zentmayer performed has been very successful in clearing the cornea, and I think he is to be congratulated upon the success of his treatment.

Dr. Langdon: I should like to ask what factor it was that made Dr. Zentmayer decide on iridectomy if there was no increase in tension. Probably it was purely a nutritional iridectomy.

Dr. Zentmayer (in closing): In answer to Dr. Langdon's question as to the indication for iridectomy in the absence of increased tension, to use Dr. Langdon's term, it was done for a nutritional purpose, and in that sense was purely an empirical procedure.

A New Technique for the Preparation of Pathological Specimens for Microscopic Study

Perce DeLong, M.D.: The method which I present has, to me, been the most satisfactory of the numerous ones employed, first, because of the saving in time by which a globe can be sectioned and presented for study in not longer than three or four weeks; second, because the sections can be routinely cut at 7 mu. (a decided advantage), and third, because the cell blocks do no have to be kept moist with alcohol solutions but can be filed away as the ordinary paraffin block.

The above advantages demonstrate the outstanding merit of this method.

Discussion. Dr. Fry: I think the one point that is to be emphasized in regard to this method is the greater rapidity with which specimens can be prepared, because by other methods it is possible to get very fine sections. Within the last several years Dr. Custer, in the Pathological Department of the University, has been using the usual celloidin method, as the result of which he is able to cut specimens as thin as 2 microns; but, in order to do that, he uses five different celloidin solutions and allows the specimens to remain in each solution at least a week. Thorough dehydration must precede the celloidin impregnation. By Dr. DeLong's method, you can get equally thin sections in one-third the time in preparation.

Report Upon 100 Intracapsular Cataract Extractions by the Knapp Method

Leighton F. Appleman, M.D., reported his results of 100 cases of intracapsular cataract extraction at the Wills Hospital, from March, 1929, to February, 1932, which were successfully accomplished out of 180 eyes operated upon.

Out of the 100 cases there were 6 failures to obtain visual results, in 3 because of retinal detachment, 2 because of subsequent iridocyclitis, and 1 because of illiteracy could not give accurate information for record. Of the remaining 94 cases, 63, or 67.02 per cent obtained 6/12 vision or better.

Of the 80 other cases of the series the lens was delivered by the usual classical method, and of these an accurate record was kept of 63. Of these, 37, or 58.73 per cent obtained 6/12 vision or better.

The time of stay in the hospital was distinctly less after operation

upon the intracapsular cases than in those delivered by the usual method.

Dr. Appleman stated that he was greatly encouraged to continue this type of operation in uncomplicated senile cataract as he felt that it is the best method to employ, since it relieves the patient of his trouble in one operation, at the same time offering one of the best chances for restoration of vision.

Discussion. Dr. Peter: Dr. Appleman is to be congratulated not only upon the results but especially upon his courage to develop this type of operation, which promises to be the operation of the future. It is a question as to what method of intracapsular extraction will eventually prevail. The Smith method is a simple one to follow when one finds himself in the position in which he cannot deliver in capsule or by any other method. One disadvantage I find in the Smith extraction is the drawn-up or hammock-shaped pupil which usually follows the extraction by this method. It will occur, particularly if the Smith hook is used too vigorously in delivering the lens. The Barraquer method will probably be the method of choice in the hands of men who do a good deal of eye surgery. The use of the capsule forceps will probably be practised by a great majority of men. My own personal experience with these three methods leads me to believe that the Barraquer method is best. One must select patients very carefully. Young patients with immature cataract are better operated by one of the three smooth forceps. For patients sixty years or older, it is safe to use a suction apparatus. Insofar as the percentage of success is concerned, in my own practice, I find about 70 per cent can be delivered by the forceps operation. The capsule ruptures in about 30 per cent of cases. With the Barraquer method, the percentage of success is much better.

If one is careful in applying the crisophake squarely to the lens, he rarely fails to deliver the lens if the patient is sixty years of age or older. I am not sure that it cannot be used on younger patients, but I have not used it on young patients. Instead I use the forcept method. Even complicated cataracts, which occur, as a rule, in young individuals, people forty-five to fifty years of age, can be delivered quite successfully with the forceps method. I do not quite see the advantage of releasing one's grasp of the lens after the zonular fibers are ruptured. When the lower part of the lens reaches the upper part of the wound, there is every reason why one should continue to keep his grasp on the lens, because the lens tumbles. One can help along the delivery by means of the Smith hook, and if little traction is exerted with the forceps themselves, the lens capsule will not rupture in delivery. I find it ruptures occasionally before delivery when the bite is too wide. For that reason, the Verhoeff forceps is perhaps the best type. The Elschnig forceps are fairly good, but a bit too clumsy. I began with the Kalt model, but feel that the Verhoeff model insures a better bite. In addition, Verhoeff's model, when properly made, is almost fool proof, in that one is not apt to squeeze too tightly and take a bite out of the capsule. In sclerosed and hypermature cataracts, it is difficult to get a bite with

forceps. In these types, the Barraquer method is much the better one. One can obtain a very good grip upon the sclerosed lens with an erisophake when you cannot get a good bite with the forceps.

So far as Dr. Appleman's statistics are concerned, they speak for themselves. The results in private patients are better than those operated on in hospitals. The latter group include all types—the Negro who is apprehensive, foreigners who do not understand our language, etc. For this type of patient, the percentage of successes reported is excellent. If Dr. Appleman will compare a like number of patients selected from private practice, his results will be even better.

Dr. George Cross: I, too, wish to congratulate Dr. Appleman on his excellent results with this operation, and will limit my comment to this type of intracapsular operation.

The method Dr. Appleman uses is similar to the technique I employ, which closely follows that of Dr. Arnold Knapp, who kindly permitted me to observe his work on several occasions. One difficulty in attempting to rupture the suspensory ligament with Kalt forceps was the tendency to pull the lens toward the incision which often ruptures the ligament. This was corrected by downward and forward traction against the counter pressure on the outside and ruptures the ligament below, as was desired.

I was interested to hear Dr. Appleman state he had four patients who had injured the operated eye during sleep. Some operators do not feel that any protective dressing is necessary, I never perform an extraction in capsule that I do not use an aluminum shield for three days after the operation, until the first dressing, and sometimes for a few days longer, so that no accident may occur.

Dr. Knapp states "that in selecting cases for the intracapsular operation it is not advisable to choose patients with a deep anterior chamber, high myopia or very prominent or protruding eyes." To me a most interesting step in the operation is the application of the forceps and their manipulation. It seems that some failures in properly getting hold of the capsule are due to the fact that the incision is not made sufficiently large. In this operation it is essential to enlarge the incision so that capsule forceps can reach over the center of the curve of the lens, without too much trauma to the cornea.

Dr. Langdon: I know most of these cases come in in such a condition that refraction cannot be studied before operation, but I was wondering, in relation to Dr. Appleman's retinal detachments, whether, since those cases with high myopia are much more prone to retinal detachment than any other, he had data on these 3 cases.

Dr. Shannon: In connection with Dr. Appleman's interesting paper, I should like to report the complication of choroidal detachment occurring in a patient eight days after a successful intracapsular extraction. The detachment appeared to be spontaneous in character. Reattachment, I am glad to state, took place three weeks later, with satisfactory recovery of vision.

May I, at this time, thank Dr. Appleman for helping me to overcome two difficulties which I had been experiencing in the technique of the intra-

capsular extraction. In the introduction of the Kalt forceps, I did not seem to secure a satisfactory view of the anterior capsule of the lens. This was obviated, as Dr. Appleman suggested, by standing to the side of the patient instead of directly behind.

My second difficulty was in determining whether the zonule had been ruptured or not. Dr. Appleman advised that the lens be drawn up sufficiently to permit the lower edge to appear above the margin of the pupil. By following this suggestion I found that a satisfactory rupture of the zonule generally occurred.

I might add that I have found it helpful to secure a full dilatation of the pupil by injecting 4 m of a 1 to 1000 adrenalin chloride solution subconjunctivally about fifteen minutes before the operation. The dilatation is maintained throughout the operation and, in addition, tends to lessen the bleeding into the anterior chamber which is most annoying at times, as we all know. Dr. Migitot carried out this procedure routinely in all his cataract cases and it was from him that I received this idea.

Dr. Harrison: We have had, I believe, 14 Barraquer operations since June, 1931. Of these, 10 have shown 20/20 vision, one 20/50 and the other 3 were 20/30 or 20/40. We had trouble in one case, with the loss of a drop or two of vitreous, due to the fact that we were too anxious to get the lens out. Most of the cases were selective and I hope some time to be able to give you a more detailed report.

We think we have had very good results and feel that later on we can probably give you a series which may in a small way reflect Professor Barraquer's ability to do the operation and to show it to others.

As to the fact of the operation being confined to those definitely skilled in this sort of work, I may say what my experience has been limited. When I arrived in Barcelona I was quite apprehensive as to what I could do. I visited Professor Barraquer for two months, saw a total of 15 cases, then he gave me my first opportunity to operate. The operation came along very successfully, and I then regularly assisted him. I do not believe it is more difficult to do than with the Knapp forceps. I have had some experience with these forceps and some with the classical operation and do not believe the Barraquer suction operation presents a more difficult task.

Dr. Appleman (in closing): In regard to this operation, I think a great deal of the success of the operation depends upon the patient himself. As you know, any considerable movement may spoil the result. I believe that a certain amount of preliminary training on the day before, directions that they may sleep over and think over, can be profitably employed. In my experience, at least, they come to operation in a much better frame of mind and coöperate better.

Regarding Dr. Langdon's question as to myopia. Ordinarily detachment did not occur in myopic cases. As Dr. Knapp has said, intracapsular operation is a little more difficult in myopia, I had 1 case in which there was no difficulty, although a few floating vitreous opacities were present and the eyeballs rather prominent, the lens was successfully removed without any deleterious happening; but here, again, it was simply a question of coöperation upon the part of the patient.

As to Dr. Shannon's knowing whether the zonule had broken, in all my cases the lens edge is made to present in the pupillary space, and remain there, otherwise I am not sure that the zonule has ruptured, by making pressure below and counter-pressure downward from above it may be tumbled without difficulty.

DECEMBER 15

Fracture of Maxilla Through Left Optic Foramen. Reduction with a Kingsley Splint with Restoration of Vision

H. Maxwell Langdon, M.D., and (by invitation) James E. Aiguier, D.D.S. Miss H. M., aged twenty-three years, after a motor accident was admitted to the hospital with a fracture of the skull running through the left orbit involving the optic foramen and sloping down on either side in the molar region. There was also a fracture in the right molar region, a fracture of the nose over the bridge and through the ethmoid sinus. There was mild shock, temperature of 95. The eyelids were swollen. When this swelling went down it was found that the right eye was uninjured, the vision normal; but the left eye had very little light reaction of the pupil and vision was diminished to perception of light and no fundus changes.

In six days there was a beginning pallor of the left optic disk. Dr. Aiguier made a splint with a plaster of Paris cap for traction. This was put in place over the teeth and pressure was gradually applied for fear that greater damage might be done to the optic nerve. As the parts adjusted themselves, there was, however, immediate improvement in vision and this continued gradually until within two weeks it had become 5/45; and in six weeks was 5/12. With a large paracentral scotoma which was partly absolute and partly relative.

The loss of vision might have been the result of hemorrhage into the sheath of the nerve, but in view of the immediate improvement on the application of the splint it seems fair to suppose that direct pressure on the nerve played part in the production of the loss of vision.

Discussion. Dr. Edward A. Shumway: From the character of the visual field, which shows a full peripheral field, indicating the absence of any disturbance of the peripheral fibers of the nerve, and as the important

part, a large partly absolute, and partly relative, scotoma situated below the center of vision, it would appear that the true cause of the visual disturbance was not a fracture into the optic canal, but rather a hemorrhage pressing on the upper part of the optic nerve, and in that way, involving particularly the papillo-macular fibers, which are well known to be more sensitive than the other fibers of the nerve. It would also be interesting to study the color fields, as my experience has shown at times marked disturbance of the color-carrying fibers in such injuries, when the nerve had become quite pale but the ultimate vision and form field had not been affected.

Dr. McAndrews: I believe Dr. Langdon stated that as soon as the splint was applied, the vision improved. Would not this tend to indicate that the bony displacement rather than hemorrhage was causing the poor vision?

Dr. Langdon (in closing): This was the point which made us believe that probably displacement of the bone had something to do with the diminution of vision. We rather hesitated about increasing the pressure by the splint as we were afraid of doing greater damage than had already been done, but instead of being diminished, the vision was greatly improved by the application of pressure. Just how much hemorrhage had to do with it, is almost impossible to tell.

In reply to Dr. Holloway's question as to whether x-ray showed fracture of the sphenoid sinus, it showed fracture through the ethmoid but I do not think the sphenoid was mentioned in the report.

Extraction within the Capsule of the Crystalline Lens Containing a Foreign Body. With the Report of Three Cases

Burton Chance, M.D., in exhibiting some lenses containing foreign bodies, briefly reviewed the subject of the management of cataract caused by the lodging of foreign bodies in the lens.

The specimens were removed entire without rupture of the capsule. His method has been to dislocate the lens, and with pressure from below to extract the whole lenticular body through the incision made as in the ordinary cataract operation. In each of the cases no complication arose, the healing was prompt and the visual result satisfactory.

Remarks Concerning Superficial Punctate Keratitis and the Associated Conjunctivitis

G. E. deSchweinitz, M.D.: After a brief reference to the textbook descriptions of superficial punctate keratitis and also to the marked clinical variations to which the disease is liable, Dr. deSchweinitz confined his further remarks to only one phase of this

subject, namely, mistakes in diagnosis, or rather, to a delay in ascertaining that a persisting low-grade conjunctivitis or conjunctival hyperemia, devoid of etiologically active bacteria, may in a good many cases be part of the symptomatology of superficial punctate keratitis or one of its varieties.

He pointed out how well this matter had been described by Col. Robert Wright in his admirable study of this disease in India, where it exists in an epidemic form.

The corneal lesions are readily detected after vital staining with fluorescin with the corneal microscope and slit-lamp, or, although not so strikingly, with a good corneal loup.

He did not think that the various forms of superficial punctate keratitis or epithelial punctate keratitis (Koppe) were more frequent now, *i. e.*, within the last ten or twelve years, than formerly, but only that they were (or should be) more often detected, owing to the increasing use of the slit-lamp, a point emphasized by Meesman.

Dr. deSchweinitz classified his cases as follows: (*a*) unilateral, with only very fine punctiform lesions, subject to relapses, first one and then the other eye; (*b*) both eyes affected, mixed lesions varying in size from a pin-point to a pin-head; also hook-shaped lesions; (*c*) unilateral or bilateral, with a tendency for the foci to form rows in addition to their scattered situation.

Generally the lesions avoided the corneal periphery; direct vision was unaffected unless large lesions were implanted at the corneal center.

These observations are not, of course, new, having been made by a number of observers, but Dr. deSchweinitz again called attention to them and emphasized them, because in a fair number of patients who had come under observation during the past few years a persisting conjunctival hyperemia had been treated for weeks, even months (in one case) without relief by the usual collyria and local applications before the corneal complication had been detected by the method described.

Dr. deSchweinitz recommended as the best lotion physiological salt solution, all irritating lotions to be avoided; a 1 per cent solution of phenacain (holocain); a 1 to 1000 oily suspension of acriflavin had proved to be useful.

Focal infections should be eliminated; in one of the cases removal of the tonsils was helpful, and in 2 others the treatment of a chronic ethmoiditis.

Because superficial punctate keratitis, or something closely allied to it, can also develop during the exacerbation period of iritis, trachoma, etc., Koby suggests it should be regarded as a morphological conception.

Discussion. Dr. Hunter W. Scarlett: Since Dr. deSchweinitz drew my attention to the close association between superficial punctate keratitis and conjunctivitis of long standing, I have been on the lookout for such cases. During the past six months I have examined 8 patients with this condition, 6 of whom were female and 2 male. Their ages varied from eight to fifty, average length of time between the onset of symptoms to the first examination by me, ranged from one week to several weeks or months. The convalescence lasted from five to six days to several weeks or months, depending on the severity of the attack, 7 of the 8 cases were bilateral, 1 unilateral, which findings were contrary to the findings of Colonel Wright that bilateral cases are not often observed.

The pathological factors were the ethmoids in 5 cases, infected tonsils in 2 and infected teeth in 1. One case of chronic ethmoiditis was complicated with impetigo.

In all cases improvement followed our attack on the foci of infection. Relief was afforded by mild eye lotions such as suggested by Dr. deSchweinitz.

Dr. Burton Chance: I am sure that Dr. deSchweinitz can recall that Dr. Norris pointed out to him, just as he had remarked to me, as far back as 1896, that, not infrequently one might find superficial affections of the cornea in cases of low grade chronic conjunctivitis which fail to yield to the ordinary remedies. Such lesions could be studied even at that early date by the simple forms of focal illumination available at that time.

Dr. Edward A. Shumway: In my experience unilateral persistent chronic types of conjunctivitis with corneal involvement are more liable to be associated with ethmoid infection, although at times the only factor found is infection of the tonsils. Where the tonsils were involved the condition is more apt to be bilateral, but in any case an exhaustive study of the entire body for foci of infection should be made in every case.

Dr. T. B. Holloway: The superficial lesions to which Dr. deSchweinitz has called attention are not only extremely interesting, but at times difficult to properly classify. The term might be applied to a group of manifestations and it is awfully difficult to exclude certain punctate lesions that at times are associated with tiny vesicle formations. Two of the most typical cases of the old type of superficial punctate keratitis that I have seen were associated with uterine conditions and both responded after several weeks of local treatment and the correction of the uterine trouble. At the present time I have under my care a patient with a low grade conjunctivitis who on the second visit presented typical punctate lesions in the cornea, followed by a series of minute vesicles. These subsided rather promptly under treatment. Aside from these there is another type of punctate corneal lesion usually located near the limbus, and in my experience usually associated with some infection about the head or with arthritic manifestation

or with a myalgia. Dr. deSchweinitz did not allude to the corneal sensation in certain of these cases. I feel that a certain number of them are definitely neuropathic and at times there is an alteration in the sensation of the cornea, in my experience the sensation is more frequently decreased.

As to the use of salt solution, one patient now under observation would use nothing else but this solution, and such has been my experience in certain other cases. In addition to this, in many of these cases a bandage is invaluable and the most important of the various therapeutic measures that could be used, but it was necessary that the dressing beneath the bandage should be thick so as to completely immobilize the lid.

Metastatic Carcinoma of the Choroid

Dr. Perce DeLong: Though many cases of metastatic carcinoma of the choroid have been reported, its rarity warrants the citation of any additional authentic cases. I therefore add the following case to the list already published.

For extensive and detailed study of this condition I would refer the reader to the articles of Marshall, Parsons, LaGrange and Oatman. In the works published in 1932, 130 cases have been reported. These, with my case and cases reported by American authors which were not included in the above total, bring the number to 135.

It is difficult to obtain accurate statistics of this disease for it is reasonable to suppose that some patients in the last stages of cachexia, die without the ocular lesions being recognized.

This discussion is restricted to specific cases of metastatic carcinoma of the choroid, and all carcinomatous involvements of the choroid as yet reported have been metastatic, but it must be stated that several cases of primary carcinoma of the iris and ciliary body have been reported. The usual seat of the primary growth (in the order of their frequency) is breast, lungs, stomach, liver, mediastinal glands and the thyroid. Rarer locations are the suprarenal body, prostate and ovaries.

The case which I had the opportunity of studying pathologically was admitted to the Lankenau Hospital, July 19, 1932, white, female, aged forty-eight years. She complained of a "boring" pain in the right eye, disturbance of vision and distortion of objects, also light flashes.

General examination was negative for heart, kidneys and lungs. The abdomen was soft and relaxed, and no tenderness or mass was

detected. A later examination revealed a nodular mass in the upper right abdomen, under or attached to the liver.

There was some exophthalmus in the right eye. The pupils were equal and reacted to light and accommodation. The sclera was somewhat injected. Fundus examination was unsatisfactory. The left eye revealed normal eye grounds. The clinical diagnosis was choroidal sarcoma. The eye was enucleated and the patient was discharged July 25.

She returned six days later complaining of severe "stabbing" pain over the right lower ribs. She had no cough; she had had dyspnea since July 26, and became exhausted after speaking long sentences. *X*-ray examination of her chest revealed a moderate bilateral diaphragmatic lag. There was a slight haziness, left base. No evidence of demonstrable sarcoma. She was discharged August 26, and died two months later. Macroscopic examination. The specimen, a globe of normal size, upon section revealed a shallow anterior chamber. In the posterior portion there was a neoplasm, flat in shape, of which the greater anterior posterior diameter was 3 mm. It terminated rather abruptly at the equator. The overlying retina was detached by a subretinal serous exudate. This separated the retina from the neoplasm.

Microscopic Examination: The preëquatorial section of the globe showed slight evidence of edema with a secondary glaucoma, manifested by a small ciliary body and processes, which were dragged forward. There was an almost complete absence of the anterior chamber and complete obliteration of the filtration angle. The retina was detached and showed evidences of disorganization and early edema.

The neoplasm was composed of epithelial cells, having a tubular or adenoid arrangement. The arrangement of the epithelial cells was rather irregular in certain areas, while in other areas they had a cuboidal outline. Other cells appear globular in shape. There was no areas of necrosis or hemorrhages. The advancing cells could be seen infiltrating in the lymph spaces of the choroid.

The stroma was composed of connective tissue with branching tuberculi. The choroidal vessels were distorted but were present in the stroma. There was some new vessel formation. Bruch's membrane was intact. The sclera was not infiltrated. There was no metastasis to the optic nerve.

Discussion. Dr. Edward A. Shumway: I think that Dr. DeLong's technique in the cutting of sections is a remarkable one, and greatly to the credit of himself and the Wills Hospital, where it was worked out. The seemingly impossible task of making sections of the entire eyeball at a thickness of 7 micro-millimeters which he has achieved is the best that I have seen.

I wish to corroborate Dr. DeLong's findings as to the carcinomatous character of the metastases in the choroid. At the time of the original examination, which I had the opportunity of seeing the patient, it was thought that the condition was a sarcoma of the choroid and later when the mass appeared in the liver, it was believed that this was a metastasis from the sarcoma. Microscopic examination of the specimen, however, showed the true condition to be a metastasis from a glandular carcinoma of the intestine, with secondary involvement of the liver. These metastases enter the eye, as Dr. DeLong has said, through the short, posterior ciliary vessels, and occasionally metastases can be found in the ocular muscles in the orbit, which were also supplied by the ciliary branches of the ophthalmic artery.

Dr. H. Maxwell Langdon: Through the kindness of Dr. Pfahler, I saw a case of his last summer, which presented a picture which was probably metastatic carcinoma of the eye. A woman, aged thirty-seven years, with carcinoma of the breast, who did not report for examination until the condition had become inoperable. She was sent to Dr. Pfahler for radiation, and the cells being amenable to the ray, the progress in the breast seemed to be checked and she seemed to be in good condition.

After about four or five months she began to have blurring of the right eye and I saw her and found she had detachment of the retina near the macula, giving a sharply defined paracentral scotoma, vision being reduced to 5/45. Of course, no pathological study was possible, but it was presumed to be a metastasis from the growth. She was sent back to Dr. Pfahler for radiation of the eye which was carried out on three different occasions. I did not see her again for six weeks when she came in with normal vision, the retina entirely in place, scotoma entirely gone. I have not seen her for about six weeks, but at that time corrected vision was normal and the eye perfectly quiescent.

I believe that this was a metastasis from the tumor growth in the breast. What the future will be, no one can tell, but that she will have a secondary metastasis elsewhere seems likely.

Sodium Carbonate (Monohydrated) in the Treatment of Vernal Conjunctivitis

Louis Lehrfeld, M.D. (by invitation): Sodium carbonate monohydrate, 5 and 10 grains to the ounce of water, is advocated in the treatment of the acute stage of vernal conjunctivitis. The directions for its use are as follows:

The eyes are flushed with cold saturated boric acid solution, seven times daily, using an eye cup; sodium carbonate is then instilled in the eyes, 3 drops, four times daily, the boric acid flush always preceding the use of sodium carbonate.

The principal underlying the sodium carbonate treatment is that this chemical actually disintegrates the stringy mucus which is characteristic of the palpebral form of vernal conjunctivitis.

The strength of the sodium carbonate is determined by the quantity necessary to partially disintegrate the mucus and to loosen it from its attachment to the conjunctiva. The flushings of cold boric acid solution serve to wash away the loosened mucus, and the cold lotion takes the place of adrenalin for its astringent and soothing effect.

The treatment, as outlined, removed the exciting agents of the eye responsible for vernal conjunctivitis, thus relieving the patient of symptoms and encouraging the recession of the pathological lesions characteristic of this disease.

Discussion. Dr. Burton Chance: Dr. Lehrfeld should be congratulated for his persistence in the study of vernal conjunctivitis. His own interest in the cases reporting at the Wills Hospital, all of which have been assigned to Dr. Lehrfeld, has afforded him the very unusual opportunity to see a larger number than otherwise he could have seen. In my own cases I use the form of treatment adopted, and I can confirm what Dr. Lehrfeld has said. Some patients come in with huge strings of gummy material in the conjunctival sacs, but these are promptly swept away by the solutions of sodium carbonate which Dr. Lehrfeld recommends.

I have used sodium carbonate in profuse conjunctivitis for a number of years. I stumbled on it by accident. I use in my sterilizer, the ordinary sodium carbonate. One day a patient came in with a greatly inflamed eye and copious discharge, I simply sucked up in a pipette, a quantity of water from the warm sterilizer, and washed the lids, to my amazement they became clean, and the patient relieved of his discomfort. Since that time I have been using sodium carbonate in my ordinary cases.

Dr. Edward A. Shumway: The question as to whether intense itching in cases of vernal conjunctivitis was due to the presence of stringly mucus is by no means established, the itching is just as intense in the limbal type, or in the milder lid types. However, I corroborate the effectiveness of a 1 per cent solution of sodium carbonate which I have used at the suggestion of Dr. Lehrfeld, and it appears to be an important observation, as it might be equally effective in the treatment of sinus inflammation, in the presence of profuse glairy, muco-pus in the sinuses, and inflammations of the bladder. It is well known that the special group of bacteria under the name of Bacillus mucosus capsulatus is capable of producing a glairy mucus. These are related to the so-called Ozena bacilli and to Friedlander's bacilli.

According to Axenfeld these organisms are an occasional find in conjunctivitis, and where such organisms existed, the secretion caused by them might well be influenced by sodium carbonate solution.

The frequent loss of the lashes in the use of radium, in the first cases treated by radium, had been one of the chief objections to the method. Since the use of a newer technique, adopted by Dr. Robert Shoemaker at the Lankenau Hospital, this difficulty has been removed, and its effectiveness depends upon the method of filtration of the radium. At present Dr. Shoemaker is employing 35 mg., in a tube, with a filter composed of silver 0.5 mm. brass 1 mm. and rubber 2 mm. Direct application to the skin surface of the upper eyelid is made with a closed eye for a period of forty-five minutes. This dosage had proved sufficient after an average number of from four to six applications at intervals of ten days to three weeks, to effect pronounced improvement, and believed to be well below the threshold of injury to the lens, which is the part of the eye most likely to be adversely affected by radiation. With this technique reactions are very mild, there is no marked erythema of the skin, and so far no loss of eye lashes in any of the cases treated.

Dr. T. B. Holloway: I arise to make these comments. First, to ask Dr. Lehrfeld what influence he though liquid albolene would have if he believed the condition caused by direct contact of the pollen. Second, to state that there is another condition that produces a secretion that has the same characteristics to which Dr. Lehrfeld referred, namely, a superficial affection of the cornea and conjunctiva probably neuropathic, it occurs at times in certain cases of severe arthritis. Third, as the result of a typographical error I was quoted at a previous meeting as using "fibrin vaccine" in such cases, what I really said was fibrolysin.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

JANUARY 20

The Anatomy of the Paranasal Sinuses in Children

Dr. J. Parsons Schaeffer: There is not much that is new to present. It is well to have facts that are not entirely new presented at meetings in order that we do not become confused. There is such a vast amount of material available on this subject that it is difficult to select the most important items. The secondary sinuses are old phylogenetically. They appear as early as the ninetieth day in the fetus. It has been frequently stated that the sphenoidal sinus does not appear until near the age of puberty. All the sinuses begin to appear about the hundredth day of fetal life and then expand. At birth the ethmoidal sinus is of clinical value, the sphenoidal sinus may be as large as a pea, and while the frontal sinus as such is not in position, the antra are already present. At this time there is a tendency, in so far as the meatuses are concerned, to expansion of the nasal fossæ for extension of the olfactory area. This function being almost lost in man, considerable may be learned from the study of the olfactory physiology in lower animals. The entire paranasal sinus series are ontogenetically ethmoidal cells, and all are outgrowths of the ethmoidal cells and communicate in the adult with the ethmoidal field. The anatomical, embryological and grosser morphological facts have been fairly well established in the United States. We have almost reached finality. In the way of physiology much is to be learned. In 1907, working on sheep, I desired to watch ciliary action. I found the olfactory area without motility and that all motion occurred toward the pharynx. Other workers have recently found that higher anthropoids, even under general anesthesia, maintain

ciliary action. These studies are in the direction future sinus work should take.

Looking at the early ethmoidal field we notice in the mesenchyme areas in which cartilage and later bone appear in the cell matrix, and that the cartilage bears a certain relation to the mucosa. If the general mass is tumefied and highly vascular we may wonder if the child will not react differently. There is no frank symptomatology in early childhood. We need to know more about the evagination of these sacs which are so important developmentally. Approaching the seventh, eighth and ninth years, the maxillary sinus does not go out into the lateral reaches of the maxilla and the frontal sinus is not extensive. They are of early importance, but we must look for a different symptomatology in early childhood.

In examining the primitive nasal fossa, no entoderm is found forward in this position in the embryo. Very soon the development of the future nares is suggested by the appearance of slight depressions, and olfactory pits, one on each side of the frontonasal process. At this time there is no evidence whatever of the sinuses except a bulge that represents the future turbinate, a proliferation of the mesenchyme, and containing only soft tissue. Later the inferior turbinate becomes well established and high up we have the first evidence of an ethmoturbinal structure. Then the middle nasal meatus, which later develops the nasal sinuses, may be observed in the precartilage stage. The hard palate is completed well forward and by the fiftieth day we find the ethmoidal field well differentiated and the conchæ developed. The secondary conchæ appear and the meati which later become sinuses. Cross-section of the forty-nine-day fetus shows the precartilage stage almost running into the cartilaginous period and further differentiation. By the hundred and tenth day the secondary conchæ are definitely present, as well as the uncinate and bulla. The maxillary sinus is a conspicuous structure and is in relation with the inferior meatus. The ethmoidal infundibulum is blind anteriorly and has often been erroneously labeled maxillary sinus. These secondary furrows are often confused. At the hundred and twentieth day we find the point of evagination of the ethmoidal infundibulum. The floor of the infundibulum extends outward to become the maxillary sinus. Sometimes the early pouching is double, which may lead to a double maxillary sinus, each cavity having its own ostium into the ethmoidal infundibulum. We have seen several cases of double

maxillary sinus, each with its own ostium. A posterior ethmoidal cell extending downward into the maxilla is frequently found and simulates a double maxillary sinus, but genetically it is not. These are very common, and their reason for existing may be demonstrated in the embryo.

In children the maxillary sinus is narrow from side to side, and this is important clinically since in puncturing the sinus it is relatively easy to enter the orbit. Up to the second year the maxillary sinus is medial to the infraorbital nerve. In the adult we find wide infraorbital contact and palatal contact. Passage into the orbit upon puncture is now almost unthinkable. In the seven-year-old child the maxillary sinus has gone to the level of the floor of the nose, is somewhat widened and its contact with the lateral nasal wall is more extensive. The ethmoidal cells are now conspicuous. At eight years the sphenoidal sinus is large, but shows no lateral growth. The ethmoidal labyrinth is developed and the frontal sinus has grown well above the nasion, but no wide recesses have appeared as yet. All of the sinuses at this time are very regular.

The relation of the milk teeth to the rudimentary maxillary sinus suggests to us the reason for lodging of the posterior teeth so that they never eruct. The measurements of the maxillary sinus increase from birth to the twelfth year.

In the six- or seven-month fetus there is noticed an open lateral recess of the middle meatus, and here are found the frontal furrows which are the progenitors of the anterior ethmoidal cells and the frontal sinus. At seven months the differentiation continues. The frontal recess as a whole moves toward the frontal region. We may have three frontal sinuses because three ethmoidal cells grew into the frontal bone, due to either unusual vascularity or heredity. In a study of 800 cases, 8 per cent were found to have a straight naso-frontal duct because continuous with the ethmoidal infundibulum.

From the roentgenographic point of view, the anterior ethmoidal labyrinth must be considered with the frontal sinus in the first few months after birth. There is some doubt whether complete absence of the frontal sinus ever occurs. Perhaps very rarely the condition may exist. There is a gradual growth of ethmoidal cells from birth to fourteen years of age. The sixteen-month infant shows a differentiation into posterior, middle and anterior ethmoidal groups. At this time the sphenoidal sinus has not yet invaded the bony bulk,

but at eighteen months some development of the bones of Bertin are developing and at five years they are still free, but have approached their permanent shape of hollow cones. The hollowing-out of the body of the sphenoid now begins, and at seven years the sphenoidal sinus is expanding. In the adult the sphenoidal sinus has extended to the greater wing of the sphenoid and bears important relations to the internal carotid artery, optic nerve and other structures. At nine years of age the sphenoidal sinus has almost reached the adult phase except for the expansion into the lateral structures. Ethmoidal cells in turbinates are merely the outgrowths of furrows.

Ciliated epithelium occurs in the nasal fossæ about the fourth month and by the fifth month is general. Glands are more numerous at the ostia of the maxillary sinuses than anywhere else. In the maxillary sinus of the dog we find cells which are probably associated with olfaction. Some cells found in the antral mucous membrane are believed to deal with dynamics. Cryptlike depression, goblet cells and more deeply placed glands are all characteristic of antral mucosa. The mucosa of the frontal sinus shows no differentiation; there are no glands and few bloodvessels. In comparing the mucosa of the maxillary and other accessory sinuses with turbinal mucosa there is a striking contrast. Many glands are present in the turbinal mucosa, while few glands are present in the sinus mucosa.

Discussion. Dr. S. R. Skillern: The hiatus semilunaris ends blindly in an anterior ethmoidal cell and not in the frontal sinus. It is contiguous but not continuous with the frontal sinus. The probe must be bent into an U shape and lifted forward and upward into the frontal sinus. I have one specimen which shows a double maxillary sinus. A supraorbital ethmoidal cell is frequently found, and by going through this at operation we may get into orbital fat or other ethmoidal cells. The sphenethmoidal cell described is a most common finding at operation. A great many eye conditions attributed to sphenoidal sinus infections are due to infections of the posterior ethmoidal cells. I would not hesitate to open any normal sphenoidal, as no resultant harm would follow.

Sinusitis in Children

Dr. Warren B. Davis: From earliest infancy paranasal pneumatization is sufficient to make sinusitis a condition to be considered clinically, not only in cases with readily demonstrable infections, but also to be investigated in many cases with obscure symptoms

and uncertain etiology. Sinusitis continues to be a most interesting and facinating field for investigation because the anatomy of the areas shows such variations, the symptomatology is so inconstant and often misleading in diagnosis and the selection of plans of treatment calls for exercise of the soundest judgment and experience in determining whether a case should be treated by nonsurgical measures alone, or, if surgical measures are also indicated, the type and extent of the necessary operative procedures.

In average cases all of the sinuses have assumed their approximate adult type of outlines by the fourteenth year, though their ultimate maximum dimensions have not been reached. Any condition which interferes with free nasal ventilation and drainage acts as a predisposing factor in sinus infections. Among such causes most frequently encountered are hypertrophic nasal and pharyngeal mucosa resulting from enlarged infected tonsils and adenoids, pale boggy mucosa associated with allergic conditions, enlarged or malformed turbinates, septal deviations, ridges or spurs, cleft-palate, atresia of nares, traumatic deformities of nose, transient swellings from trauma, foreign body in nasal passage and obstructing tumors. Among general predisposing causes are endocrine disorders, metabolic disturbances and lowered resistance from unbalanced or deficient diets. Not infrequently sinus infections develop as complications or as sequellæ to acute colds, tonsillitis, measles, scarlet fever, whooping-cough, pneumonia and more rarely diphtheria. Lack of sunshine, unfavorable climatic conditions, especially excessive dampness and sudden changes in temperature, swimming and diving are etiologic factors in other cases. There is scarcely another location, with the possible exception of the tonsils, in which infection is known to produce such a wide range of symptoms, complications and sequellæ. To enumerate some of the conditions in which we know that sinus infections must be considered when endeavoring to locate the primary underlying cause, the following list is believed to be a practical one for clinical purposes: Acute or chronic pharyngitis with congestion extending downward from the nasopharynx along the posterior faucial pillars, hypertrophy of lymphoid follicles in the posterior pharyngeal wall, submaxillary adenitis, cervical adenitis, frequent colds, recurring epistaxis, otitis media, phlyctenular conjunctivitis, orbital cellulitis, orbital abscess, optic neuritis, persistent cough without demonstrable intrathoracic cause, acute or chronic bronchitis, bronchiec-

tasis, enlarged peribronchial lymph nodes, peribronchial thickening, nontuberculous pulmonary infiltration, anemia, anorexia, malnutrition, asthma, myocarditis, chorea, acute rheumatic fever, arthritis, recurring fever of unknown origin, nephritis, pyelitis, meningitis, cavernous sinus thrombosis and brain abscess.

The diagnosis of sinusitis undoubtedly is made more frequently now than during previous years, but it is probable that the apparent increase in the number of cases is due to more careful diagnosis, both by pediatricians and rhinologists, rather than to an actual increase in the relative frequency of the infection. In this series the relative frequency with which the predominating infections have been located in the different sinuses of children is, in descending order, ethmoidal, maxillary, sphenoidal and frontal. Ethmoidal infections often occur alone. Maxillary infections are nearly always secondary to ethmoidal infections, but may sometimes persist alone after the ethmoiditis has subsided. In patients under fourteen years of age maxillary sinusitis of dental origin has not been encountered. Sphenoidal infections in children always accompanies a posterior ethmoiditis, the ethmoiditis most probably being the the primary infection. Frontal infections in childhood are always secondary to anterior ethmoiditis and are treated as ethmoidal infections. No cases of frontal sinusitis in patients under fourteen years of age were seen in which radical operative measures were felt to be necessary.

While the majority of sinus infections in children occur between the fourth and ninth years, definite virulent infections are not infrequently found in infants. A case of suppurative maxillary sinusitis in an infant, aged thirteen days, presented bone destruction which had been so rapid that spontaneous perforation of the anterior maxillary wall had already occurred. Death resulted on the nineteenth day from pyemia, multiple metastatic abscesses and septic pneumonia. Another case at six weeks of age developed an ethmoiditis resulting in death two weeks later from an orbital abscess and meningitis.

A review of the literature shows increasing stress being placed upon diet and allergic states in relation to sinus infection. In diagnosis, attention must be given to a careful history, including family tendencies. The examination must be thorough. Supraorbital pain and headache may occur, but not so frequently in adults. Transillumination is of some diagnostic value but is not as reliable

as in adults. Roentgenological films are indispensable in making an accurate and complete diagnosis. Bacteriologically the infections are usually of a mixed type, and any of the organisms frequenting the upper respiratory areas may be found. Pneumococci and streptococci usually predominate.

Treatment is more difficult in children than in adults and requires both experience and patience. The best results have been obtained by a routine which follows: (1) Regulate diet and correct nutritional disturbances so far as is possible. Many cases require cooperation with a good pediatrician. (2) If there is an allergic state, an attempt is made to identify the allergen and to eliminate its contact if possible, or else to increase immunity to it. (3) Internal medication. In acute cases small doses of atropine sulphate at three- to six-hour intervals are given, while in older children ephedrine sulphate is sometimes used instead of atropine. Calcium, with small doses of parathyroid, often is beneficial. Cod-liver oil is given once or twice daily. In subacute or chronic cases iodide of iron is used. Vaccines are valuable aids in many cases. (4) Local treatment: Mildly astringent oil solutions alternating with argyrol, 10 per cent, are preferred. Ephedrine solutions sometimes cause irritation or even superficial ulceration when used liberally. (5) Surgical procedures should be conservative. The endeavor is to secure ventilation and drainage with the least possible trauma and destruction of intranasal structures. (6) If after nonsurgical treatment polypoid tissue persists in the ethmoidal area it should be removed, also the anterior portion of the middle turbinate. (7) Many maxillary infections clear up after removal of an enlarged middle turbinate. (8) Orbital abscesses, due to extension from ethmoidal infection, most frequently form between the lamina papyracea and its relatively thick layer of fibroperiosteal covering. The accumulating pus causes separation of this covering from the bone and, associated with the surrounding cellulitis and edema, causes lateral and anterior displacement of the orbital structures. Drainage of such abscesses through the anterior ethmoidal area is most satisfactory. The middle turbinate, the anterior ethmoidal cells and a part of the lamina papyracea are removed. In all of this series of cases there has been an associated suppurative maxillary sinusitis. Thus a nasoantral opening is made beneath the lower turbinate at the same time. A supplemental external incision into the orbit is seldom necessary, but if it is believed that the

purulent exudate has extended beyond the fibroperiosteal layer an incision is made along the upper medial orbital wall and a small drain inserted. After any operation on the sinuses continuation of postoperative treatments until the areas are completely healed is important, since synechia form more readily in children than in adults.

Discussion. Dr. Philip Stout: For years I have been interested in a point which Dr. Davis has mentioned. Frequently we see dehiscences in the orbital wall and in proximity to the ethmoidal cells. These make us wonder whether orbital abscess is not more frequent than we realize. There are no lymphatics in the orbit and drainage in this field is nasalward. At the posterior portion of the nasopharynx it is possible to get into the lymphatics and inject them.

Dr. Karl M. Houser: I would like to ask Dr. Davis how he gets dissemination of colloidal silver solutions without the use of constrictors. My experience has been that a puddle forms of the colloidal silver if constriction is not obtained first. Such constricting agents as ephedrine or weak solutions of adrenalin (epinephrin) are usually employed. I have not felt that reactions occurred nor seen the ulceration mentioned. I saw a small child confined to home by scarlet fever. Thirty-six hours before there had been complete closure of the eye with chemosis and fixation of the orb. Bronchitis made the prognosis bad. Ten per cent cocaine solution was used with adrenalin and some space appeared between the middle turbinate and the septum. A rubber tube was inserted and every fifteen minutes a $\frac{1}{4}$-pipette of ephedrine sulphate was introduced into the tube. The following day there was excellent drainage from the nose and subsidence of the swelling. In another twenty-four hours the child could open its eyelids and the chemosis was almost gone.

Dr. H. A. Schatz: Sinus trouble always begins in infancy, but there may be remissions. The hereditary factor is important. Accessory ostia result from expansion from the ethmoidal area with penetration of the surrounding bony structures. The process may be likened to the bursting of bubbles, one sinus is formed instead of two. The ostium always forms in the thinnest portion of the bony wall.

Dr. James A. Babbitt: I would like the question of interference with the drum considered. In later life is there any effect on hearing function? Where radical and sphenoidal operations have been done, has anything been observed in children as to diminution of olfactory function?

Dr. Davis (in closing): Dr. Stout mentioned the use of suction. I do not use suction. I think the removal of exudate by a pipette is sufficient. Suction by a stronger apparatus often produces a degree of congestion which offsets our intention in using it. In adults we frequently use it to produce artificial hyperemia, but it is not favorable in acute conditions. In answering Dr. Houser's question, I may say that we give internal doses of atropine. With ephedrine by mouth a longer action is obtained, and doses of $\frac{1}{1000}$ grain may be used in infants. Locally the favorite solution

is: cocaine, 1 grain, and camphor, 1 grain, in liquid albolene; this mixture keeps the opening free for a longer interval. There is no objection thus far to weak solutions of ephedrine, but I have seen so many cases in recent months where this solution was used by the patient on their own responsibility that it has become a pernicious custom. In reply to Dr. Schatz, I believe a careful history, especially of allergic factors and definite family tendencies, to be most important. Whether all sinus conditions begin in childhood is difficult to say. Remarks on the developmental questions have been demonstrated by Dr. Schaeffer. I have never seen a case where the bubble-fusion effect has occurred. When double maxillary sinuses occur there is no opening in the intermaxillary septum excepting as it may occur pathologically. In the field in which we operate we do not come into the area of olfactory function.

Lymphatic Drainage From the Maxillary Sinuses

Dr. G. E. Pfahler: I have had an unusual experience and one to which I can find no reference in the literature. A man, aged thirty-two years, had had pain throughout the body for eight years. For three months he had noticed large lymph nodes in the neck and other parts of the body and there had been pains in the neck. These symptoms led to a search for foci of infection. In his home city in West Virginia, lipiodol had been injected into the maxillary sinus. Operation had been suggested but refused. A lymph node was removed from the groin and pronounced lymphosarcoma. Clinically, the case did not resemble lymphosarcoma. A review of the case, by Dr. Case, showed general inflammatory changes. There was a residual tonsillar tab, lymphangitis in the pharynx, but no other obvious lesions. The man recovered in two months' time and has been well since. In restudying the maxillary sinus, patches of lipiodol were found extending from the zygoma into the retropharyngeal space and as far as the subclavian vein. In these roentgen films and lantern slides you see the deposits extending up the side of the orbit. Plates, a few days later, showed deposits extending along what we took for lymphatic channels and clearly shown at the entrance to the thorax and from there being traced into the thoracic duct on the left side. A remnant was found in the right antrum eleven months after the injection and drainage spaces still definitely extended above the maxillary sinus and down into the thoracic duct and subclavian vein. The distribution seems to outline lymphatic structures throughout the neck. A glance at charts of lymphatic structures helped to explain

why in malignancy we get extensions upward from such regions as the parotid area.

Discussion. Dr. J. Parsons Schaeffer: This is the most interesting thing presented tonight, whether actual or accidental. The lymphatic drainage from the nose always causes discussion. Textbook figures list lymph nodes along the jugular and intercalated nodes; we find many more outlying nodes at dissection than are pictured. When lymphatic capillaries are examined they are found to be unlike blood capillaries. They start as small channels, balloon out to form lakes, narrow and again widen. Because of its weight lipiodol may have found its way out of the lymphatics into the tissue spaces. The upward trend is not to be wondered at as this is merely following the trend of the lymphatic routes. The interesting feature is that none was carried into the thorax or lung.

Dr. Philip Stout: I injected 200 antra in an allergic series and no lipiodol remained in the sinuses longer than two or two and a half weeks unless it went outside the antrum. In that event it did not spread. Lipiodol should leave the ostium in three or four days or seven or eight days at the longest.

Dr. Pfahler (in closing): I should have added that, after this experience, we reëxamined other patients without duplicating this appearance. This has lasted a year, and may last another year, before it will be absorbed. It seemed to me that presentation of this case might lead to the finding of other similar cases.

FEBRUARY 17

Sinusitis and the Rôle It Plays in the Production of Asthma

Dr. H. B. Wilmer: It is well recognized by most observers in the field of allergy that subacute and chronic infections of the upper respiratory tract, such as sinusitis, tonsillitis and adenoiditis, are important etiologic and contributory factors in the causation of asthma; but they do not all agree and are at variance to the relative importance of such factors and the mechanisms by which they operate.

It is definite and, I believe, thoroughly possible that in asthma, as well as in hay fever, nasal infection may play the rôle of an excitant as well as a complicating factor.

Quoting Gottlieb, the upper respiratory tract may cause bronchial asthma by one of the following explanations:

1. Constant drainage of pus into trachea and bronchi.
2. Toxic absorption from damming-up pus in sinuses.

3. Mechanical obstruction of air-passages, causing mouth-breathing, thereby irritating pulmonary mucous membrane.

4. Reflexly by irritation or infection of sphenopalatine ganglion.

I definitely feel that nasal obstruction with resultant mouth-breathing plays an important rôle in the etiology of asthma. Infected tonsils and adenoids are also etiology factors in asthma, but are too often blamed, and removed without justification. Intranasal and tonsil surgery should only be resorted to in an asthmatic after a careful examination has ruled out atopic hypersensitiveness.

The failures to relieve the symptoms permanently in the surgical procedure far outnumber the permanent cures. I do not believe any surgical procedure is wise during the pollination period unless extremely urgent. A potentially allergic individual may become clinically active following nasal operation during the hayfever season.

The efficacy of vaccine therapy has been discussed *pro* and *con* for many years. I personally have had more than moderate success with this form of treatment as shown by the following results: Using bacterins only, total of 141 cases; no relief, 31; improved, 73; cured, 37.

I feel that the success of bacterin therapy lies in the collection of the nasal smear, the care with which it is prepared, the method of testing and the method of administration. Each organism is isolated and cultured and the patient tested intradermally. The organisms producing immediate and delayed reactions are used in the preparation of the bacterin. Reculturing may be necessary before results are obtained.

Where there is decided infection of the nares or sinuses, bacterin therapy alone, without the aid of the otolaryngologist, is futile. Frequently a stock vaccine may be used with as much success as an autogenous preparation. It has been our procedure to give small doses of the vaccines over a long period of time. They are given at five- to seven-day intervals. Many cases will require continued administration after the initial dosage has been given, at monthly intervals, especially through the winter months, as an aid preventing relapses.

Charts: The instance of bacteria found in 682 cases of asthma: staphylococcus, 237; streptococcus, 493; pneumococcus, 350; catarrhalis, 306; diphtheroid, 118; Gram-negative bacillus (Friedländer's), 101. The instance of bacteria found in nasal smears of 122 cases

of asthma: staphylococcus, 82; streptococcus, 45; pneumococcus, 28; catarrhalis, 21; diphtheroid, 36; Gram-negative bacillus (Friedländer's), 12.

Bronchial Asthma From the Rhinologic Standpoint

Dr. Harry P. Schenck: While bronchial asthma has come to be regarded as an effect resulting from more than a single process, and many of the precipitating factors have been recognized, the actual cause of asthma remains unknown. The mechanism of the asthmatic paroxysm was described and the various possibilities of vagus stimulation discussed. Intranasal pathology may influence bronchial asthma by reflex stimulation of the vagus or by influencing bacterial sensitization. Experimental work which points to a nervous pathway from the nose to the peripheral bronchial fibers of the vagus was described, while it was emphasized that the actual pathway has never been satisfactorily traced. Bacterial allergy, while not well understood, is operative because of blood-borne protein fractions, polysaccharides or amines which may directly stimulate the vagus endings.

Allergists and rhinologists are agreed that the asthmatic patient is primarily an allergic problem. The rhinologist should enter the picture only after the completion of skin tests and either failure or unsatisfactory results from desensitization and elimination of reactors. The end-results of nasal surgery differ in various clinics, and whether to employ radical or conservative measures is still in dispute. Not every asthmatic patient has sinus disease. The development of edema of the soft tissues and polypoid degeneration with later secondary infection of the paranasal chambers does not occur suddenly. If asthma precedes extensive changes in the nasal structure the allergist is often able by the elimination of reactors and desensitization to prevent alterations in the upper respiratory tract. It is important to prevent nasal pathology as to prevent emphysema and chronic bronchitis. The future asthmatic, with present vasomotor rhinitis and pollen sensitivity, frequently consults the rhinologist first, and it is his responsibility to detect early allergic alterations and his duty to guide the patient to the allergist. Early prophylactic measures of this sort are more valuable than any amount of radical surgery in the advanced case. Early cases require no surgery and are frequently made worse by intranasal medication.

Surgery in the allergic individual is always attended by certain elements of danger. Allergic symptoms frequently follow surgical procedures. Surgery should never be performed on an allergic individual during the active pollen season. Postoperative sensitization to bedding materials may occur. The rhinologist may well blunder into increasing rather than alleviating allergic symptoms. Asthmatic patients are particularly prone to postoperative erysipelas.

The asthmatic patient may be worse after operation because: (1) Sensitization to new atopens at the time of operation or shortly thereafter; (2) reflex stimulation of the vagus by tight packing, or postoperative edema of the tissues; (3) denudation areas facilitating the absorption of allergens; (4) infection of new areas due to faulty technique, such as failing to remove packing within a reasonable time.

The asthmatic patient may be worse after operation because: (1) Most important, employing surgical methods when there is no indication for them. A submucous resection on a septum which is angular but fails to make lateral contacts is not likely to be of benefit. (2) Incomplete surgery. Removal of polyps from antrum while polyps are permitted to remain undisturbed in the other. (3) Faulty postoperative treatment. Adhesions must not be permitted to form. New tissue is subject to the same allergic influence as the diseased tissue removed, and unless we are prepared to protect the new tissue it is useless to remove the old. The rhinologist is concerned primarily in securing aëration and drainage. Local measures such as colloidal silver packs, roentgen ray, radium and physical agents were discussed. Vitamins and acid-base diets were outlined.

In surgery of these patients no cure should be promised, but remissions may usually be expected during which the patient gains weight and regains some measure of hope. Because of the necessity for multiple procedures it is imperative to secure good local anesthesia and observe the greatest delicacy in the manipulation of instruments, so that the patient may approach successive operations without fear and with confidence in the operator. The patient must be repeatedly warned that attention to diet and elimination of other reactors are important after operation and that surgery is only one line of attack. Both allergists and rhinologists are confronted by the unsolved problem of what to do about nasal path-

ology in asthmatics. Neither a *laissez faire* policy nor indiscriminate radical surgery has solved the problem, but there is definite evidence that conservatism is a safe rule.

Discussion. Dr. John A. Kolmer: I have enjoyed the two papers this evening and agree with most of the points as stated. My mind has been well made up in regard to the sinuses in allergic individuals. If skin tests are positive, then proceed without surgery; if tests are negative, then bacterial antigen is probably the cause, and sinuses should be carefully examined and if found involved, I advise surgery. Most surgical cases relapse, and the question is asked, why? I believe it is due to incomplete surgery, as removal of all infected material is almost impossible. A case of frontal sinusitis is cited that improved for two months following intranasal surgery, but had a relapse. A radical operation was done and the patient has been symptom-free for two years.

I believe that persons acquire a sensitivity to bacterial proteins and in the majority of cases the sinuses are involved.

Dr. Richard A. Kern: From what has been said it seems that we are agreed that bronchial asthma is a syndrome of symptoms. The question of which mechanism is of prime importance in asthma has been answered contradictory. The former viewpoint is wrong, due to own particular type of patients studied. Now the clinics study all cases of all ages and from every angle. The most important factor in 75 per cent of cases is hypersensitivity and 25 per cent infection and nasal reflex. Frequently the cases are a combination of the above two groups.

Just how infection complicates asthmatics is not clear. Sinus infection in this locality is common and in 72 per cent of cases, used as controls in an asthmatic study by Schenck and myself, there was found sinus infection. It was also shown that by *x*-ray the asthmatics had a smaller percentage of sinus infection. Too much stress should not be placed on the relation of sinuses to asthma.

It is hard to find a rhinologist who is willing to be radical enough to clean out everything, yet it is important to completely remove all pathological tissue. Even radical surgery is sometimes followed by poor results. I saw one case that had had a bilateral Caldwell-Luc, and a secondary operation showed more pathology in the antra than was there before. I only saw one case in which tonsillectomy helped asthma.

I believe in bacterial protein allergy, but feel that the initial dose should be extremely small.

Dr. Arthur Wagers: I have been pleased by the note of conservatism in the papers and discussion tonight. My feeling is that nasal conditions rarely cause asthma. However, a definitely pathologic sinus or irregular septum can initiate the asthmatic spasm.

Sinus *x*-ray is frequently reported negative; but there are other methods of determining if pathology is present and these should always be used in making a diagnosis. If polyps are found they should be removed. Dr. Kern's remark regarding thorough operation is timely. The ethmoids,

due to their anatomical configuration, are difficult of complete exenteration. A careful study of each individual case is most important.

Dr. Karl M. Hauser: My reaction is one of confusion, and this is due to end-results obtained. Asthma can be caused by reflex without sinus involvement or allery. Adams, of Glasgow, reports a group of asthmatics treated entirely by rearranging their diet. Asthma does not occur among the Eskimos. They are not subject to pollens, but are constantly in contact with animal emanations. All things have a bearing on asthma, but the real cause has eluded the investigation.

Dr. Gabriel Tucker: An important point in bronchial asthma that has been overlooked is the wheezing tracheobronchial tree due to spasm of bronchial muscles. After all other methods of treatment fail, patients will frequently respond to bronchoscopic treatment.

Dr. George B. Wood: Most cultures taken from the nose are contaminated. I have used a glass tube with narrowed lumen at each end connected to a suction tube. The glass tube is placed at the sinus ostium, suction applied, and when the tube is withdrawn sealing the ends. This eliminates contamination and the tube can be sent directly to the bacteriologist.

Dr. Samuel R. Skillern, Jr.: I wonder if the work begun by Stucky and now being carried on by Jarvis, in Vermont, has not hit upon the background of asthma in relation to acid-base balance of the body. The red septum is the acid type and can be controlled by an alkaline régime. The pale septum shows the body balance to be on the alkaline side and the color can be brought to the septum by administering dilute nitrohydrochloric acid. I believe all foci of infection should be removed in seeking a cure, including the removal of tonsils.

Dr. Matthew Ersner: Allergy is never found in patients with a red septum. With the pale septum the secretions from the nose are alkaline in reaction and in these cases the calcium in the gastrointestinal tract is not properly assimilated by the body. Starve the patient; then put on a milk diet; lactic acid is formed and this helps to absorb the calcium. In patients with watery nasal discharge in which eosinophils predominate allergy should be suspected.

Dr. Justin M. Waugh, Cleveland: The more you see of asthma (as a rhinologist), the less you think of yourself. It is not possible to eliminate all the infections in all the sinuses. Although our present knowledge is in a state of confusion, the succeeding research can go along more smoothly by discarding all worthless reports, just as the study of cancer is being carried on. I do not believe that dietary disasters are the whole cause of asthma or that it will eliminate asthma. I have not had Dr. Tucker's success with the bronchoscopic treatment.

Dr. Wilmer (in closing): I feel that we as allergists are very much in the dark but that we are getting results in some of our cases. I agree with Dr. Kerns in radical surgery of the sinuses when their involvement is definitely determined. There is no similarity of patients in the field of allergy and the physician must study each patient as an individual problem.

MARCH 16

Intimate Notes on the Ear, Nose and Throat Clinic

Dr. William Hewson: These remarks are a plea to consider the ear, nose and throat dispensary more seriously. There are some in the profession who might be called dispensary nihilists. They would abolish all dispensaries as unnecessary and detrimental to the best interests of the profession and laity. They base their contention on three fairly well-recognized observations:

1. The growing abuse of the free clinic.
2. Lack of coöperation between the hospital and the outside doctor.
3. The comparative small number of cases referred to the house from the dispensary.

Dispensary abuse has existed ever since the dispensary was organized, and most of us will admit that today more people who are able to pay are using the free clinic than ever before in its history. This number is increasing every year and very little if anything is done about it. It seems to be a social problem and as such is referred to social service departments and agencies. I believe the average social service department of a hospital incapable of successfully dealing with the matter because, even if there were enough workers and enough time to ask all the questions, the patients themselves would continue to falsify their answers in order to get free treatment.

Lack of coöperation between the hospital and the outside doctor has hampered the progress of both for many years. The outside doctor wonders why a patient sent to the hospital for one condition is retained by the dispensary for another. He, of course, chafes at the discovery of several of his pay patients being treated in the dispensary free. He feels ignored by the hospital, which takes his patients, sends him no word about them, perhaps operates on them without his knowledge and in the after-care gives him no hint of why the hospital wants them in the dispensary. He perhaps resents most of all the competitive spirit of the dispensary and he cannot reconcile a charitable institution underbidding him.

Cases referred to the house from the dispensary are quite few, if tonsil cases are not included. This is particularly apparent in hospitals where tonsil cases are dealt with by the receiving ward

only. Chronic ear and sinus cases make up the next largest group. These are benefited by treatments in the dispensary, but most reliance is put on the directions for home care. Dispensary visits cannot be arranged often enough for the cases because the dispensary days are too few or too crowded to be effective. It is contended that most acute cases should not be ambulant and, therefore, should not be treated in the dispensary. The "dispensary nihilist" finally calls our attention to many of our western states where the large outpatient clinic is the exception rather than the rule.

In the course of time since the organization of the dispensary we have to admit that the object of the dispensary has broadened, as well as its public acceptances. Patients no longer dread the idea of going to the hospital, if they do not have to stay. In fact, the dispensary idea has been successfully sold to them, "oversubscribed," we might say, and sold cheaply. The average dispensary is overcrowded, with long lines of waiting patients. Patients cough and sneeze; children run about and get in the way; weak patients, requiring only postoperative instructions, stand about. Contagious cases, chronic running-ear cases, foul-smelling atrophic rhinitis cases are all gathered together waiting their turn. Inside the consultation room there is almost as much of a crowd and very much confusion. There need not be detailed the various activities going on there, with each patient usually in view of others. It is worth recording, however, that the dispensary doctors are more than busy and are working under pressure.

Some wit has described the dispensary as a place where patients are dispensed with and gotten rid of in the speediest time possible. Such a dispensary may be spoken of as "going to seed." The most important sign of such a condition is lack of interest on the part of the dispensary doctors. They are perhaps overworked, the equipment has not been kept up, the contacts and encouragement from other members of the staff are wanting. It becomes easier for them to get in late and leave early. Records are poorly kept or no notes made at all. The incentive for work is lost and the morale of the dispensary suffers. Tired or cross doctors do not reflect credit on themselves or the institution. It makes very little difference how important a dispensary may be, it will lose its worthwhileness if the morale is lowered, the work becomes drudgery or the general atmosphere becomes electric and combative. It is useless to try

to interest younger men in such a dispensary, no matter how many and varied are the opportunities. Their interest will only be short-lived and we cannot blame them. In such an institution the hospital authorities are beside themselves to know what to do. The house chiefs are usually ignorant of what the situation in the dispensary really is, and they would rather no think about it. It is quite evidently nobody's business, so that the dispensary soon becomes an overcrowded torture chamber to the patient, a sweat-shop to the dispensary doctor, a sore thumb to the hospital and a neglected stepchild to the hospital staff.

The object of the up-to-date dispensary has been extended to include not only a philanthropic effort for the poor patient, but also a broadening educational opportunity for the doctor. No dispensary doctor works for nothing if he is truly interested in his work and has the opportunity and capacity for doing it well.

A suggested plan for a moderate-sized clinic would be very briefly as follows: Patients would be divided into two groups, a free clinic and pay clinic. Each would meet twice a week, Monday and Thursday or Tuesday and Friday. Both would be presided over by the same staff under the direction of the clinic chief. The free clinic would be an examination and advice clinic where very few if any treatments would be given. Doctors working on a treatment problem could, of course, have cases admitted for this purpose, but the number of such problems would be limited by the dispensary chief. Myringotomy and antrum washings would, of course, be done as well as single treatments of acute cases. An appointment system, managed by the hospital staff, would limit the number of cases to the capacity of the clinic and make it possible for both pay and free clinic patients to know within fifteen minutes of the time they could be seen, so avoiding long waiting and crowding. pay-clinic patients would be asked a fee comparable to the office charge of the outside doctor, about fifty cents. The dispensary doctor, with some appropriate title, would be on a salary paid by the hospital. His appointment for service could be for six months and he would be allowed only one other hospital appointment. A time-checking system would be adopted, so the doctor and the hospital would both be protected. Dispensary assistants, without title, would be solicited from among outside doctors, doctors on other staffs in the hospital, resident physicians and others wanting to observe a number of ear or sinus cases, etc. These would be

under the direction of the chief of clinic, but assigned to the dispensary doctors for instruction. Dispensary assistants would not be limited and not be admitted to the pay clinic. Monthly meetings would be held either separately or in conjunction with the hospital staff meetings, and one report would be required from each dispensary doctor during his term of service. Volunteer lay-workers would be assigned secretarial jobs and make certain hearing and statistical charts.

The hospital would furnish well-lighted and well-ventilated consultation and treatment rooms. These would be divided by movable screens or curtains into treatment cubicles. There would be a sufficient number of cubicles for each dispensary doctor to have two. A dark-room and a hearing-room would be conveniently located. The usual instruments would be adequate. Provision would be made to examine all children under six years of age lying down.

Discussion. Dr. James A. Babbitt: Some of the routine details mentioned in Dr. Hewson's paper may be at variance with our old ideas, but the practical points are well worth considering. It is pleasant to sit down, with plenty of time, and work on a dispensary case without competitive, social and other factors. I like the term "clinic," and am not certain that it does not properly apply to the outpatient department. Dispensaries may be the most important part of a hospital. Well-appointed facilities with close coöperation with related dispensaries are essential.

Dr. Philip Stout: The hardest problem is the pay clinic. There is no reason why every poor person should be taken to a hospital when the city government is paying doctors to do this work. The pay patient at once wants more than a student; he wants the professor, even if he is paying but fifty cents. Physicians on the outside are getting a trifle tired of hospitals taking their patients. It is difficult to separate at the door those able to pay and those not able to pay; separate the goats from the sheep; treat the sheep and send the goats home.

Dr. Matthew S. Ersner: Dr. Hewson has brought an enticing utopia before us. The drudgery of the doctors, the poor waiting patients, the crying children and the other features mentioned combine to make a gloomy picture. We all know conditions are not satisfactory. If people are taking to free treatment, the tendency is toward state medicine. If we think progress is being made toward state medicine then the profession should fortify itself so that physicians may dictate certain terms. I have put in ten or twelve years in dispensary work, but find that we hurry in our offices as well as in the dispensaries. Perhaps we should socialize medicine.

Dr. Hewson (in closing): The dispensary problem is one which we are not meeting adequately. When Dr. Ersner says we are drifting toward

state medicine he is dead right. We cannot antagonize outside physicians. The clinic must be thought of very differently than formerly. Hospital has a problem to solve in its relation to outside physicians. Dr. Babbitt mentioned the pleasure obtained in taking time on dispensary patients and thoroughly studying the case. If men working in clinics could spend time over individual cases and have apparatus adequate for study there would never be a paucity of dispensary men. In reference to Dr. Stout's remarks, there is no doubt but that the physician in present circumstances is competing with himself.

Actinomycosis of the Tongue and Tonsil

Dr. Robert F. Ridpath: After a review of the literature on actinomycosis, the case of a male, aged forty-two years, colored, was reported. The patient, when referred from the dispensary to the ward, was unable to talk and was admitted with the diagnosis of actinomycosis of the tongue and left tonsil. Eight days before admission there had been severe pain in the tongue and the left side of the face, which was attributed to an injury by a new tooth-brush two weeks previously. The physical examination was negative except for alterations in the mouth, neck, pharynx and tongue. The tongue was greatly swollen and completely filled the mouth. The left tonsil was enlarged, engorged and covered with mucus and ulcerations. Large, hard masses were noted along the left border of the tongue. The posterior cervical glands were palpable. The supraclavicular and infraclavicular fossæ were filled. Tracheotomy was performed because of the dyspnea, but the swelling continued. Eleven days after admission there was softening and contraction of the area along the border of the tongue. A spinal puncture was made and clear fluid obtained under a pressure of 12 mm. The spinal fluid Wassermann was negative. The urine was negative for iodine, although iodine was given in large doses. The typical ray fungus was isolated and granules identified. The temperature was never over 101 F. The total white cell count was 16,000 on admission, with 84 per cent polymorphonuclears, 9 per cent small lymphocytes and 7 per cent large lymphocytes. A week later the white count was 8400, with 53 per cent polymorphonuclears and 32 per cent small lymphocytes and 32 per cent large lymphocytes.

The patient received liquid diet and a carbolic mouthwash. Incision and drainage of the peritonsillar abscess was followed by tracheotomy. Sodium iodide was administered intravenously in doses of 30 grains, three times a day. Twenty days after admis-

sion the tracheotomy tube was removed and ten days later the patient was discharged. The tongue was still thick and hard, but there were no new lesions and the general health was good.

Discussion. Dr. John A. Kolmer: I have deep interest in actinomycosis of the tongue. The 2 cases of actinomycosis of the jaw which I have seen were both in farmers. This is the first case of lesions of the tongue which I have heard reported. Serum therapy, vaccine therapy and chemotherapy are of no avail. Potassium iodide is the important agent. One case received as much as 500 grains a day. Heroic doses are necessary. I have no explanation for the surprising result of urine negative for iodine under such dosage.

Studies on the Prophylaxis and Treatment of Septic Meningitis

Dr. John A. Kolmer and Dr. K. Amano (by invitation): The mortality of streptococcic and pneumococcic meningitis has stimulated the search for adequate therapy. A clinical and laboratory experience extending over a period of ten years is here summarized. The majority of cases of streptococcic and pneumococcic meningitis are secondary to otitis media or sinusitis. The organisms may enter the postnasal area and enter the meninges. Their invasion by any route presents the most important surgical problems. When there is impossibility of extirpating the focus, reinfection of the meninges from these areas occurs. Prevention has been thought possible by the administration of immune sera given preoperatively. The signs and symptoms of meningitis may develop during pneumonia, but the majority of these prove to be meningismus. The majority of recoveries reported in the literature are of this type. Streptococcic meningitis may develop during scarlet fever without ear or throat lesions. The majority, however, are secondary to primary infection of the ear, mastoid or sinuses. Surgical drainage must be established. I have never seen a staphylococcic meningitis which was not secondary, and these tend to remain localized. The signs and symptoms may be indicative of localized meningitis, but the spinal fluid may be clear early and mislead diagnosis so that meningismus is suspected. It is difficult to decide upon a radical form of treatment unless the signs and symptoms of diffuse meningitis are noted. It is impossible to save any animal if more than twenty-four to forty-eight hours have elapsed after the onset. I have never seen any harm result in withdrawing 5 cc. of spinal

fluid. There has been no dissemination of infection. The pressure is measured with a mercury manometer In septicemia no infection of spinal fluid results from lumbar puncture.

In early localized meningeal congestion pressure may or may not be increased and the spinal fluid may be clear. In localized meningitis the fluid obtained a few hours after onset shows few cells, increased protein and sometimes an increase in sugar. Absence of organisms in direct smears may be all that is lacking, but should not be permitted to give a sense of false security. The danger lies in waiting. Infection continues to spread during the remaining few days Radical surgical procedures and serum therapy should not be delayed. In specific chemotherapy there is no one specific substance. Surgical drainage and serum therapy must be relied upon. There should be complete extirpation of cells if the mastoid is involved and the best possible drainage when sinusitis exists. Wherever exposure of the dura is possible, wide excision for free drainage of spinal fluid should be made. There is a difference of opinion concerning the advisability of lowering spinal fluid pressure. Experiments of our own are not conclusive, but it is probably feasible to obtain results by draining in this fashion. If infection is progressive, more radical procedures are necessary. Spinal drainage for six to eight hours, with increase of fluids or laminectomy with forced fluids are indicated. We may even consider continuous spinal drainage. In our dogs immune sera alone failed. Sera plus drainage also failed. Stewart's method of serum with ethylhydrocuprein, when drainage was also employed, was found excellent and there were recoveries amounting to a high percentage. Type I pneumococcus in the experimental dogs was fatal in four to seven days, while the control dogs died in twenty-four to forty-eight hours. Under ether anesthesia a double frontal trephine was made and the needles introduced in each cerebral subarachnoid space. This was repeated in twenty-four hours. Many recovered permanently. Some animals treated similarly with physiological sodium chloride solution alone, recovered. The treatment in the human cases was not as effective, but all 5 cases were late and practically moribund. The subarachnoid space was obliterated by plastic exudates. In dogs there was no doubt of success because the treatment was instituted before the plastic exudates formed.

In pneumococcic and streptococcic meningitis, sera and chemical agents may be introduced into both common carotids with surpris-

ing ease and safety. There is no bleeding when the technique is mastered. Given this way there is no great toxicity of the drug, and bilateral injection may be used as well as cisternal drainage.

In dogs Felton's combined antipneumococcic serum has shown definite value, while the antistreptococcic sera have not been very effective. The Parke-Davis antistreptococcic serum has given somewhat better results than others. Of course, in these dogs there was no primary focus in the mastoid cells of sinuses and, therefore, the condition did not parallel the human cases. Of 10 dogs with streptococcic meningitis, 2 received Pragel's iodine and concentrated antistreptococcic serum by intracarotid injection. Injections of neutral acriflavine may be used with greater confidence.

In the prevention of streptococcic and pneumococcic meningitis bacterial examinations should be made both before and after operation. Specific prophylactic measures must be instituted early. Intravenous and intramuscular injections of Felton's polyvalent serum influenced the incidence of meningitis in rabbits and dogs. We should culture a sinus before operation and culture the material in a mastoid wound at operation. The oral administration of pneumococcic vaccines increases the resistance of animals to pneumococci and may raise the immunity higher than when given subcutaneously. Similar results are not obtained by the oral administration of streptococcic vaccines.

The clinical applications of this work may be summarized as follows: (1) Vaccination. Operation may be considered one week after the last dose of vaccine. In pneumococcic infections it may be better to give the vaccines orally. (2) Serum prophylaxis. (3) Type the pneumococci in such infections. (4) In Type I pneumococcus infection give 10 cc. Felton's polyvalent serum and repeat on the second and fourth days. In Types II and III pneumococcic infection the serum should be used in 15-cc. doses. The same doses may be used in children over fifteen years of age. (5) The optochin base may be given in addition to serum. This is best given in 5-grain doses in milk every five hours after operation.

Discussion. Dr. Philip Stout: There are two schools as far as this question is concerned: One waits; the other goes ahead. It is still questionable which is correct.

Dr. Matthew S. Ersner: Dr. Kolmer has brought us a valuable message. We had 2 cases at Mt. Sinai which may be of interest. The first case had meningitis following mastoiditis, showing 1800 cells in the spinal fluid. The next day 30,000 cells were noted in the spinal fluid, so the carotid was

opened and polyvalent serum and acriflavine introduced. The second case occurred four months ago and we are still waiting for secondary brain abscesses to form. It might be well to bear in mind a few precautions. All of these patients, whether meningismus or cerebral hydrorrhea, have increased intracranial tension and, therefore, should receive glucose, magnesium sulphate and plenty of spinal punctures. This should be followed up with polyvalent serum. I do not believe the sinus question can be emphasized too much. One case, starting as a pansinusitis, developed meningitis. When the sphenoid was opened sphenoiditis was found. These are best treated with much shrinking but no surgery. In one patient we found difficulty in regard to the fluid intake. As soon as the fluid intake was over 500 cc., severe headaches occurred and the visual fields became contracted. I am interested in the possibility of administering vaccines by mouth. In 1916 Coates and I read a paper describing various methods of administering vaccines, but our results were not gratifying, and I hope these newer methods will prove satisfactory.

Dr. William Hewson: The question of whether we are dealing with a case of meningismus or one of meningitis is always a bugbear. A boy, aged six years, at the Abington Hospital, gave a history of ethmoiditis on the right side, which was treated in the hospital. The boy was sent home, but returned later in convulsions. The ethmoidal area was exenterated at once and intranasal maxillary drainage performed. The meningeal irritation continued for four days. Later it was learned that an otitis media had occurred four months previously. The drum at this time was doughy and, although it was incised, nothing was recovered. Four days after paracentesis the ears began to discharge and mastoiditis developed. Type III pneumococci were isolated from the wound. The spinal fluid showed a pressure of 40 mm. and 400 cells. The patient was normal after mastoid operation.

Dr. Samuel R. Skillern: The pneumococcus is the organism that has a preference for bone. We are glad to know how to govern meningitis because then we will be able to govern osteomyelitis. I have frequently called in Dr. Kolmer on these cases. In a child with plastic exudate, seen a short time ago, we were too late. How shall we know when to go in and when not to go in? We frequently see a postoperative rise in temperature. If we wait forty-eight hours we may be too late.

APRIL 20

Some Experience in the Treatment of Bilateral Abductor Paralysis

Dr. W. B. Hoover, Boston (by invitation): A study of 19 cases in the Lahey Clinic was the basis of this report on bilateral abductor paralysis. The cause of the paralysis in 18 cases was related to thyroid conditions. The clinical history in cases of bilateral abduc-

tor paralysis following thyroid operations varies. After the operation the patient may be hoarse, unable to speak above a whisper, breathing may be quiet or stridor may be so marked that tracheotomy is necessary. Of the 3 reported cases of bilateral abductor paralysis proving temporary in this clinic, tracheotomy was necessary in 2; and in the cases of bilateral abductor paralysis produced elsewhere, 2 had tracheotomies immediately following thyroid operation. Usually there is a fair airway and breathing is not difficult. With time, the voice improves and stridor develops. The glottis becomes so narrow that the activity is limited by want of air. In one of the reported series stridor began and the voice returned two years after thyroid operation. With the stridor a crowing noise develops on inspiration, especially when the patient is asleep, and on exertion. Breathing is made more difficult by respiratory infections. There is also difficulty in swallowing, some patients being unable to eat at the table with the rest of the family on account of "choking spells," believed due to the aspiration of food or secretion into the larynx or trachea.

Examination of the patient shows the signs of inspiratory obstruction, while expiration is not difficult. A view of the larynx reveals the arytenoids and vocal cords in the midline, with only a narrow chink between them. During expiration the cords are lifted upward and are opened in the midportion by the current of air and they are drawn downward, and together in a valvelike manner, as pointed out by Dr. Jackson, closing the already narrow glottis. With the history and examination the diagnosis is really made. Then comes the question of treatment. A perfect answer to this question has not yet been given. The ideal method of restoring function to the paralyzed muscles of the larynx by nerve anastomosis seemed possible, but the clinical reports of Dr. Frazier and our experience seem discouraging. Cutting of the recurrent nerve has been advocated to produce the cadaveric position of the vocal cords. In the cases mentioned this procedure was followed by no particular change.

It is well to consider here the position of paralyzed vocal cords. One might expect the cadaveric position of an arytenoid and cord in a recent complete paralysis of a recurrent laryngeal nerve. As time goes on it is reasonable to expect the paralyzed cord to approach the midline because: (1) There is no resistance to motion toward the midline; (2) it is pushed toward the median line by every act

of deglutition which closes the glottis, and the arytenoid and cord are pulled by the opposite side when only one nerve is paralyzed; (3) the soft tissue of the larynx, between the cord and cartilage, may offer resistance to the arytenoid returning laterally. In partial loss of function of the recurrent nerve, if Semon's law is true, again one would expect the vocal cord and arytenoid to be found in the median position. This was true in 2 of the cases of temporary abductor paralysis (bilateral) produced in the clinic. Experience shows that the median position is more frequently found than the cadaveric in cases of paralysis of the vocal cords.

Widening of the anterior portion of the larynx by plastic operations is mentioned and we regret trying it in a single case. Lateral transplantation of the anterior attachment of the vocal cords, as suggested by Trotter and Moore, was tried in 1 case and is mentioned only to be condemned. A number of methods with the purpose of producing the abductor position of the arytenoid, by suturing the cord to the thyroid cartilage by resecting a window in the cartilage with the expectation of the cord being drawn laterally by scar tissue, and by the implantation of the omohyoid muscle into the cord, and direct transplantation of a nerve into the posterior muscle, have all been advocated, and nearly all have been complete failures.

A great many other methods have been described but tracheotomy is one of the oldest but best methods of treatment. Here an adequate airway may be easily obtained, and if combined with a valve tube as that designed by Dr. Tucker, an excellent voice is also retained, without inconvenience to its possessor. If the patient is happy with the tube, no further treatment is necessary. A tracheotomy should be done whenever the activity of the patient is limited by the narrowed glottis, and should not be removed until the obstruction is relieved. Some patients have detested the tube and demanded further procedures.

Submucous resection of the vocal cord has been described with very promising results and such a method used in 4 cases is presented for consideration. This procedure should be done when the patient is dissatisfied with a tube, and when recovery of function is no longer probable, that is, nine months to one year after operation on the thyroid. The patient must not be promised a good voice, but told there is reason to believe there will be a whispered voice that will be easily understood, and it may be even better than

the buccal voice of a laryngectomized patient. The size of the breathing space depends upon the individual's occupation. Roughly speaking, the voice will be diminished as the airway is increased, unless the false cords can be brought together on phonation, forming a pseudoglottis. Where the valve action of the cords previously referred to is marked, a small change may make improvement in the airway if it puts the valve out of order.

A female patient, aged thirty-five years, came to the clinic with the chief complaint of dyspnea dating from an operation for hyperthyroidism sixteen months prior to admission. With the return of the voice seven months after operation there was difficulty in breathing and swallowing. For nine months there had been stridulous breathing. Four weeks before admission tracheotomy was necessary. Examination showed the arytenoids and the vocal cords lying almost in the median line. A direct laryngeal examination was made and with some difficulty the vocal cords and the arytenoids were pried apart. A catheter was placed through the larynx into the trachea enabling the patient to breathe with ease and under local anesthesia a tracheotomy was done. The catheter was removed and later a laryngofissure was done. One blade of a scissors was introduced into the opening and carried upward between the vocal cords. The thyroid cartilage and mucous membrane of the larynx were opened in the midline. A long gauze pack was placed in the upper part of the trachea above the cannula to prevent the aspiration of blood or secretions. Silkworm-gut sutures were placed in the upper and lower portions of the thyroid cartilage to serve as retractors and were held by forceps. With sharp dissection the mucous membrane was separated from the underlying structure below the true cord, over the cord and upward behind the ventricle of the larynx. The separation was carried posteriorly behind the vocal process of the arytenoids. The soft tissue was then separated from the undersurface of the thyroid cartilage. With curved scissors, the posterior attachment of the loosened tissue was cut. The vocal process of the arytenoid was removed with punch forceps. One bleeder was caught and controlled by diathermy. The loose membrane was sutured anteriorly to the cartilage with absorbable catgut. The wick in the trachea was removed. A small iodoform gauze pack was then placed in position, holding the membrane against the cartilage, filling the lumen of the larynx. The end of the iodoform pack was brought through the cricothyroid

membrane; the cartilage, subcutaneous tissue and skin were brought together and sutured. Eleven days after operation the pack was removed from the larynx and two days later the tracheal tube was plugged and the tube removed on the following day. The post-operative course was all that could be desired.

The advantage of this operation is that the mass of the cord is removed and the mucous membrane lining the larynx remains, preventing the formation of granulation and secondary cicatricial contractures. Laryngofissure today is a relatively safe operation, and an excellent exposure is attainable. The only disadvantage is the loss of voice, which is not complete.

Discussion. Dr. Louis H. Clerf: I want to congratulate Dr. Hoover on his able presentation and excellent contribution to the relief of this distressing condition. These cases are all cases of impending suffocation which are fortunately not often fatal. I have often observed this difficulty in swallowing and there is probably some sensory disturbance back of it. One case of bilateral paralysis after thyroidectomy could swallow little but fluids and took mostly milk so that scurvy later developed. Esophagoscopy was done but no real obvious cause for the difficulty in swallowing was found. The tips of the arytenoids were found tilted forward in this case. As to treatment, if nerve anastomosis is necessary, it is first of all necessary to remove obstruction. Tracheotomy is not popular because of the tube, but once done it is difficult to get the patient to part with the tube because of the ease of respirtion. I would like to ask Dr. Hoover if he has been troubled by granulations along the anterior commissure. I have treated 4 patients with an endoscopic method in an attempt to remove a margin of vocal cord throughout the whole length as well as the vocal process. In 3 cases successful results were obtained and a fourth case was operated upon yesterday. During the process of healing, granulations proved embarrassing.

Dr. Gabriel Tucker: Dr. Hoover's new operation promises to be a great advance in the treatment of this condition. Three of the important functions of the larynx are the respiratory, the protective and the phonatory. The most important to life is the respiratory. The next most important is the protective function. The production of voice is very important but not a necessary factor in the life of the individual. In bilateral posticus paralysis, the respiratory function is most impaired; the voice is less impaired. When the patient becomes urgently dyspneic, the protective function is also impaired because the secretion is aspirated into the air-passages in the effort to get air into the lungs. In restoring to normal the respiration and protection to the lower air-passages, the ideal procedure is anastomosis of the nerve. As yet that procedure has not obtained ideal results, although Dr. Charles Frazier has made very great advances in this work. Operations on the vocal cords themselves may restore the respiratory function without impairing the protective function, but there

is a definite impairment of the voice after practically all of the operations. Dr. Hoover's operation would be ideal if it could supply sufficient innervation to allow the patient to develop a voice. In observing the patients on whom Dr. Frazier has done an anastomosis there seems to be an increase in the muscular actions that produce adduction following the anastomosis. I have often felt that such an operation as Dr. Hoover has devised might give a result that would take care of all three functions of the larynx in the cases that have had an anastomosis. Dr. Clerf's suggestion of shaving off the margins of the cord certainly deserves consideration as it is a procedure that does not require external operation, not even tracheotomy in some cases, and it should not give rise to any serious scar formation within the larynx. I agree with both Dr. Hoover and Dr. Clerf that as long as the patient is satisfied with a tracheotomy tube it is the best thing, probably, to let them alone. The valvular tube takes care of the patient's difficulty in breathing, overcomes the tendency to aspiration and aids them materially in phonation. The objection to tracheotomy has been raised that the air has not been made warm and moist before it passes into the lung. It is debatable whether or not this procedure has any bad effect. The patient's own objection to wearing a tracheotomy tube must be considered, although it should not be given too much weight if the larynx is carrying out its functions properly with the use of the tube.

Dr. Hoover (in closing): I have here a cast of a larynx showing the details of the procedure. In regard to aspiration and difficulty of swallowing, I believe the effort is due to difficulty in getting air into the larynx and, therefore, there is difficulty in keeping secretion out of the larynx. Granulations occurred in only one case in which I broke through the membrane. I have not attempted the removal of enlargements of the cord because the end-results as reported in the literature seemed unsatisfactory. Even if this method failed sufficient mucous membrane may be left. Once these patients are asleep, they make a roaring noise which may be heard three or four rooms away. I have found Dr. Tucker's tube very satisfactory, as it permits the secretion to be coughed from the throat to the mouth and expectorated.

The Otological Problem Presented by Suppuration of the Petrous Pyramid

Dr. Samuel J. Kopetzky, New York (by invitation): The problems presented by suppuration of the petrosal pyramid are rather simple when basic principles are considered. It is not something new, not a new conception, but is a reassembling of data which have found their way into the literature. It may be recognized, handled and cured, or it may produce chronic otorrhea or, more to be deplored, result in meningitis and death. In the literature these cases have appeared as special reports. We have less and less of the atypical to discuss when we comprehend fundamental path-

ology. In those studies of cases coming to autopsy with Gradenigo's syndrome it was found there were meningitides. A condition which could be recognized long before Gradenigo's syndrome appeared was overlooked. We must first of all discard Gradenigo's syndrome as connected with the petrous pyramid. We may have Gradenigo's syndrome without petrosal suppuration and we may have petrosal suppuration without Gradenigo's syndrome. Osteomyelitis of the pyramid, osteitis of the petrosal pyramid, etc., have been confused and added to our misunderstanding. It is necessary to separate the pathological entities. We must accept Wittmaack's theory of of pneumatization of the temporal bone. We must go back to Wittmaack and then we will have an explanation for what happens. The temporal bone was intended to be pneumatized but it is not always pneumatized. Where there is no pneumatization we can have no mastoiditis and where there is mastoiditis there must be pneumatization. The process of pneumatization takes place not only in the mastoid process but also in the petrosal pyramid. The osteitis reaches the meninges by lymph channels and vessels. The advance is slow. Only occasionally is it acute and then becomes the more acute as the endocranial structures are reached. The difference between osteomyelitis and empyema must be recognized because differing treatment is indicated.

When osteomyelitis goes through the Haversian canal system, we are apt to get subperiosteal collections of pus and extradural collections of pus. When the lesions reach the endocranium the process goes up to the tentorium and meningitis occurs. In the osteomyelitis of the petrosal pyramid sequestra form but often in empyema of the coalesced tip no sequestra are formed. The advance in coalesced tip follows the same route as in coalescence of acute mastoiditis. Theoretically we can get a coalescence of a pneumatized tip without mastoiditis.

One group of acute mastoiditis cases fail to be aided by mastoid operation and these have been called Wittmaack's acute mastoiditis. No mastoid surgery is adequate to eliminate infection when the tip of the pyramid is involved and the ear continues to discharge. Chronicity does occur in those mastoids with pneumatization. Chronic cases that subsided if studied from the tip angle will show coalescence. The other result of suppurative lesions in the petrosa is meningitis. These cases run their course and suddenly "fulminating" meningitis occurs, so-called in comparison with men-

ingitis following osteomyelitis. Lipiodol injected in these cases went into the middle fossa and spread immediately backward and was held by the tentorium.

When the mastoiditis exists there develops after a period of time a clinical picture which is simple. There are four periods: (1) Eye pain and aural discharge; (2) low-grade sepsis; (3) quiescence; (4) onset of meningitis.

Eye pain occurs on the same side as the lesion and is in the distribution of the first branch of the fifth nerve. This must be differentiated from lateral sinus thrombosis and posterior fossa lesions. Every effort to account for the pain should be made before ascribing it to first branch pain. The onset may be from eighteen days to seven months. The aural discharge either fails to dry up or the discharge is accompanied by the eye pain. The discharge varies from a few days to months after operation. Only in the acute necrotic mastoiditis of Wittmaack can this occur in a pneumatized mastoid. The middle-ear when opened at operation is full of detritus, which should not be present and the appearance is not what we expect. During the first period there are transient signs, vertigo, nystagmus (due to labyrinthitis) tests show labyrinth functioning normally and the labyrinth is simply transiently irritated. The facial paralysis is transient.

The period of low-grade sepsis shows an afternoon temperature of 99° to 102° or 103° F. Just then the patient is reported perfectly well. There is no more facial weakness, no more transient labyrinthine irritation. This is the worst and most dangerous period. Rupture and entrance into the endocranium have occurred. It is the calm before the storm. It is almost too late now. We must fight before they reach the quiescent stage. Once in the quiescent stage there is positive roentgenological evidence. The fourth stage represents the onset of meningitis. We all know the details, opisthotonos, Kernig's sign and the others. The laboratory data is mostly negative excepting for the *x*-ray studies. The blood studies are not significant except in the terminal stage. The spinal fluid and the eyeground studies are likewise not significant except in the terminal stage. The roentgenograms give important data. Every one must get used to one roentgenologist doing his roentgenology. We want to know the presence or absence of pneumatization. As a routine, we take key pictures of the base in every mastoid case. That key plate compared with plates at the second or third stage

of the condition will prove invaluable. Is it or is it not a pneumatized tip?

There is only one treatment, operative. While suggestions have suddenly cropped out everywhere almost at the same time, surgical treatment has been agreed upon. We must differentiate the case first; then we must know what to do. In dealing with tip suppurations, the literature teems with suggestions. A few have reported cures.

The technique is simple but sounds difficult. A simple mastoidectomy has usually been done before the lesion develops. This is converted into a radical operation, the zygoma is then rimmed off almost to the glenoid articulation to get as far forward toward the Eustachian orifice as possible. There is no fear of facial palsy since Duel's work has become known. Remove the tubotympanic cells completely and remove the tensor tympani, bore into that area and open. When the patient is lying on the table, a probe in the external auditory canal gives the general direction. Open the tube and go in at an angle of 30 degrees.

In conclusion, if you will take Gradenigo's syndrome as a cross-country hunter takes a fence—take it in passing and pass on—and diagnose the lesion you are dealing with and apply specific surgery to the specific lesion, then you will be faultless and the results will be gratifying. The taking of key plates is important.

Discussion. Dr. Oscar V. Batson: It is difficult to discuss a beautiful presentation such as this one when one is not acquainted with the patients clinically. I believe it is well to forget the Gradenigo syndrome because there are only two nerves (the seventh and eighth) in the temporal bone that may be involved. Other nerves are not directly in the bone and pus is in the ramifications of the temporal bone and, therefore, cannot involve them directly. Many times there are phenomena of bloodvessels about the nerves, and I do not believe it possible for pus to lie directly against the nerve. Recent work shows the dura mater to be a part of the cranium When there is involvement of the dura there is difficulty at once, but once the process reaches the inside of the dura there is immediate trouble. At what age does pneumatization of the tip of the temporal bone appear? I have seen many bones opened in the laboratory. In the case of a child, aged eight years, peritubal cells were involved and pneumatization extended beneath the superior semicircular canal. In subacute cases the fossa may sometimes be traced out into the mastoid area below the superior canal. It may extend to the dura without involving any cells.

Dr. Francis C. Grant: The important points that I have gleaned from Dr. Kopetzky's presentation are: (1) cases occur with pus at the tip of the temporal bone; (2) a method has been evolved to drain these cases

and get them well; (3) without surgery, meningitis and brain abscess are inevitable. By this method, it is possible to localize and evacuate the pus and thus is a step in advance of older methods. If following a mastoid, meningitis develops, death results. Brain abscess is more favorable but the idea I have gathered is that here we have a condition in which a prophylactic measure is offered by which the lesion may be localized and drained. These conditions must be recognized and the technique mastered.

Dr. Matthew S. Ersner: Dr. Kopetzky's presentation tonight has cleared up many points formerly vague to me. The form of treatment depends upon whether or not pneumatization has occurred. This is a real message. I will review a few clinical symptoms to which Dr. Kopetzky has called our attention. First of all we must be keen observers to note the labyrinthine symptoms and the fleeting palsies. What should we do at the quiescent stage? I have a patient now at this stage. This talk makes me feel like rushing off and operating at once. We may have a chronic suppurative otitis media and the patient may recover. What damage can we do if we operate, providing we know our anatomy? None. We do a radical mastoidectomy, remove the tensor tympani muscle and with a drill attempt to drain pus. If no pus is found we have performed a good radical mastoidectomy and no damage is done. When I first heard Dr. Kopetzky last year I had some doubts but when I stressed some of these things in my own mind I became convinced. As to laboratory aid in analyzing cases, eosinophilia was found in 4 cases. In infection the eosinophilia disappears but reappears after infection disappears. In bone destruction and in allergy we have eosinophilia. A case of otitis media neonatorum which had lasted a week or ten days was roentgenographed and mastoid cells were demonstrated. I would like Dr. Kopetzky to discuss the Vidian nerve.

Dr. Edward B. Gleason: I have listened with rapt attention to the magnificent address made tonight. In the Graduate School I have done 48 Kopetzky operations on cadavers. There was great difficulty in getting together the specimens but I have brought some of these specimens with me. Out of 48 specimens, there was only one that showed the pneumatization that Dr. Kopetzky has described. The only case I have operated upon was a case of that kind and it made a very good recovery.

Dr. Samuel Bricker: Having had the most recent case in the literature, I want to bring out a few points. We are apt to get a definite picture before going ahead. My case had supraorbital and retroöcular pain. I was certain of petrous tip suppuration and had read Dr. Kopetzky's literature, but I did not find aural discharge and there was no septic temperature. This subsided and on the eighteenth day there was a sudden feeling of wellbeing. A roentgenogram was made but we did not know just what to look for, but there was a difference between the two petrous tips. We could not conscientiously operate until the eighteenth day. The discharge then changed from thick, sparse to mucoid secretion.

Dr. Kopetzky (in closing): I wish that I had the time and you had the patience for the elaboration of all the questions brought up tonight. I do not know how early pneumatization occurs. Thousands of cases probably do not have it. At the fifth to sixth year we begin to have pneu-

matization and the youngest of my cases was five years of age. Observation on these cases requires many times the effort the average mastoid case requires. They take seven or eight visits a day. I have been through this argument with consultants and families and for twenty-four hours have argued continuously until the family and consultant conceded the point and the patient lived. I have one case which recurred after two years. The temperature was 99° to 100° F., with fifth nerve pain and discharge from the ear. A Yankauer probe was pushed through the wound and the patient got well. The nasal sinuses must be ruled out and we must have a clear-cut case before proceeding. This is an entity and you will meet it. There is no fixed routine by which these cells develop.

OCTOBER 19

The Nature of the Static Labyrinth as Determined by Experiments in Amphibia

Dr. William J. McNally (by invitation): As we have long known, sensory hair cells, similar in general structure to those found in the cristæ of the ampullary sacs, are found in the utriculus and sacculus. Each collection of hair cells, together with the supporting cells, is designated as a macula, the macula utriculi being found in the utriculus and the macula sacculi being found in the sacculus. Lying among the hairs of the hair cell are found masses of small crystals of calcium carbonate, the otoliths or otoconia. The utricle and saccule in man have two otoliths, but in animals there are frequently more otoliths.

The kinetic function arises from the semicircular canals, while the otoliths serve as a static mechanism. The usual conception of utricular function is that it is concerned with posture. These organs are assumed to give us information regarding the position of the head when at rest and when making progressive, that is, nonrotatory movements, supplementing, therefore, the functions of the semicircular canals on the supposition that these latter act especially in movements of rotation. According to this view, the otoliths act as a means of mechanical stimulation of the hairs. Being heavier than the endolymph, they press upon the hairs with a force varying with the position of the head and thus give rise to

sensations adapted to the maintenance of equilibrium. A sort of continuous discharge of such reflexes is assumed to occur from the contact of the otoliths with the hair cells in the dynamic organ of equilibration.

If both labyrinths are removed from a frog, it may assume normal or unusual positions, but it maintains its position in spite of the lack of labyrinths and, therefore, the position is due to a neuromuscular mechanism. Change is the essence of stimulation. If the utricle is responding to a steady pressure, then it is the only known sensory epithelium that is so acting.

When a normal frog is placed upon a plane and the plane is tipped slowly so that the movement is below the threshold of stimulation of the canals, the frog responds by discontinuous shifts. If the utricle were steadily stimulated the shift would be steady and not discontinuous.

If we now remove one labyrinth from a frog, the animal leans to one side. By manipulating such a frog and bringing its posture to the midline the frog maintains its new symmetrical position when released. The moment the head is jarred or moved, however, the frog regains the asymmetrical position. A decerebrate frog is normal in labyrinthine movements, but voluntary movements are curtailed. It is the necessary jar of the utricular otolith that causes response. There is no steady pressure, but there is a sudden stimulus.

By removing all six canals in a frog, only the utricules remain as active vestibular end-organs. The posture is normal at rest, but any spontaneous movement causes the animal to sway and the jumps are not straight. If we now remove the utricles as well, the swaying immediately disappears. The canal response is so rapid, that response occurs in 1/25,000 of a second.

In the frog, maintenance of position is evidently a muscular affair. When the canals are present, they act to keep the head level and prevent the sway which the utricles would otherwise bring about. The static mechanism is evidently muscular.

Discussion. Dr. B. H. Shuster: I do not know whether the conclusions are correct or not as far as this experiment is concerned, but this work in neuroötology is of great importance. Dr. McNally's work has been brilliantly planned and directed with practical applications in mind. In showing the anterior semicircular canal controls certain muscles, the neuromuscular mechanism may be histologically proven.

Pulmonary Complications of Tonsillectomy, with Report of Three Cases

Dr. James A. Babbitt: Hemorrhage, anesthetic shock and pulmonary involvement are the three grave complications which may follow the operation for the removal of tonsils and adenoids. It would be natural to assume that pulmonary complications occurred through some carelessness in routine or predisposing pathology. Yet this has happened after thirty years of active service in connection with the tonsil operation in several hospitals, with scarcely a case of definite pulmonary sequence—3 cases have occurred in services under the writer's direction, 2 of lung abscesses and 1 of pneumonia, in the past two and one half months, and these have followed what were reported as perfectly normal tonsillectomy and adenoidectomy operations.

Operations were done in each case after routine reference from the outpatient department, with the customary preliminary safeguards performed in each case by a different operator, under ether anesthesia, in the prone position and with the use of the suction tube.

Case I.—C. M., a boy, aged three years, was admitted to the Children's Hospital August 8, 1932, for tonsil and adenoid operation, and sent home the following day. On August 11, 1932 he was brought to the medical dispensary with fever of 103°. Four days later he returned to the nose and throat dispensary. The throat was about normal, but the left ear was injected. Four days later he returned again, the left tympanic membrane was incised, the right was noted as dull and he was admitted to the hospital with a temperature of 100°. Both tympanic membranes were incised August 20, 1932, but the temperature continued to fluctuate between 100° and 103°. Blood studies showed red cells just below 4,000,000 and white cells between 11,200 and 10,300, with 70 to 72 per cent polymorphonuclears The patient developed a cough and foul breath. The mastoid and sinus roentgenograms were negative. The sputum examination showed mostly hemolytic streptococci. A roentgenogram of the chest revealed a large, dense area filling the upper lobe and a circular layer within this reaching from the second to the fourth rib, due either to abscess or cavity. Medical consultation found bronchovesicular breathing with occasional râles in the right chest anteriorly. Bronchoscopic drainage was begun and cultures of the bronchial secretion showed predominence of hemolytic pneumococci with a moderate amount of nonhemolytic streptococci and other strains. Biweekly bronchoscopic treatment and postural drainage were given by Dr. Gabriel Tucker. The left mastoid was suspicious, but both ears have cleared and the child is practically well.

Case II.—E. D., a girl, aged five years, was operated at the Mary Drexel Hospital for hypertrophied and diseased tonsils on September 8, 1932. Slight bleeding from the left tonsil required one suture. The day following operation the temperature rose to 102°. This continued irregular for a week, from 100° to 101°, then went to 104° five days later. Crisis occurred the next day. The child coughed up much mucus and the temperature gradusally dropped to normal. Nose, throat and ears appeared negative the first week. On the ninth day fine râles were heard at the left base and the next day diagnosis of bronchopneumonia was made. Roentgen examination showed marked infiltration at the roots of both lungs, clouding of the left lower and lower half of the left upper lobe. No cough or other subjective symptom was noted until the ninth day. Blood examination was conventional with the highest leukocyte count 16,000. Sputum examination showed, among other flora, a few fusiform bacilli and spirilla. Final roentgenographic examination on September 30, 1932, showed thickened pleura and some infiltration at the root of the left lung but no consolidation or fluid. The child was discharged on September 30, 1932, in good condition.

Case III.—C. A., a girl, aged three years, with chronic nasal discharge and septic tonsils, was admitted to the Children's Hospital and tonsils and adenoids were removed September 9, 1932. She was seen three days later in the nose and throat dispensary and the throat was healing well. She was brought to the medical dispensary two days later with a temperature of 101°. Two days later she was admitted to the hospital with a temperature of 100.8°. The chest examination showed slight limitation of respiration and increased tactile fremitus over the right upper lobe, impaired resonance with coarse bronchial râles over both sides. For a few days chest signs increased, with difficulty in coughing-up mucus, but the temperature remained about 102°. The first roentgenogram on September 15, 1932, showed an area of mottled density with poorly defined borders in the fifth extending to the sixth interspace posteriorly on the right side. This was diagnosed as lobar pneumonia or beginning lung abscess. The diaphragm on the right side was elevated, but the heart and trachea were in normal position. A roentgenogram the next day showed widespread, finely mottled infiltration, scattered throughout the right lung and to a lesser extent in the left. This suggested tuberculosis. Four days later, infiltration was found scattered throughout both lungs, more marked in the right. The diagnosis was in doubt between tuberculosis, bronchopneumonia and multiple small abscesses. The child has been improving under treatment and a later roentgenogram, October 4, 1932, showed diminution of the mottled area. The condition presented the initial flare, if not the actually pulmonary abscess, with a tubercular background. Bronchoscopic drainage has been delayed.

Discussion. Dr. Gabriel Tucker: Only one important point concerns the bronchoscopist in these cases and that is the matter of treatment. It is imperative that diagnosis be made early and that bronchoscopic treatment be instituted as soon as it is indicated. This type of therapy must be emphasized because it is the only effective means of combating the condition.

Dr. George M. Coates: I think it is an excellent thing to bring these accidents to our attention and make us more careful. I would like to ask Dr. Babbitt to what he attributes these abscesses. I would also like to ask Dr. Tucker whether he, as a bronchoscopist, sees many of these cases following tonsillectomy. I suspect many of these cases are not getting into the literature.

Dr. Benjamin D. Parish: Usually, these cases occur once in 2000 or 3000 cases, and here Dr. Babbitt runs into several cases in a short time. In the experimental production of lung abscesses in dogs, the condition appears to be the result of embolism plus infection. The jugular is tied off and the clot infected. The septic clot is then injected in the opposite jugular and, infection being present, lung abscess will result. Even after local anesthesia, the bronchoscopist can demonstrate some blood in the tracheal tract. I was unfortunate enough to meet one of these cases some years ago and the patient died, refusing to go to the hospital.

Dr. George Wood: I have had 1 case. The child got well with bronchoscopic treatment by Dr. Tucker. It was an inspiratory infection. A small plug was coughed up a few days later, followed by the discharge of pus. The clot was made up of clotted blood and milk. I experimented on dogs, but could not produce lung abscesses in the dog, while rabbits developed abscesses if a mere shot was introduced into the lung.

Dr. Louis H. Clerf: The aspiratory theory has the strongest evidence in support. In 77 cases we found 73 had been operated under general anesthesia and 4 under local. Only 10 per cent occurred between infancy and ten years of age. The upper-lobe lesions were more common than the lower-lobe lesions. Depth of anesthesia is probably the important thing.

Dr. Karl M. Houser: I had one experience four or five years ago. A girl, twenty-five years of age, was operated under local anesthesia. Two days after her discharge from the hospital, she developed pain in her chest and a dry cough. The temperature was 104°. The roentgenologist reported a lung abscess in the left upper lobe. There was no expectoration. Expectant treatment alone was used and the temperature dropped to normal and at the end of two weeks the patient went home. At the end of three years a chest roentgenogram was negative. This was probably an embolus, but fortunately was not a septic embolus.

Dr. William Moore: It cannot be definitely settled just how these things occur. What interests me is what we can do to avoid such happenings. We concluded that the most important thing in an adult was not to give morphine. The reflexes are then present at the time you need them. In our query, the reply was invariably that morphine had been given. To operate with the head low helps somewhat, and the after care also helps.

Dr. Matthew Ersner: I am glad that Dr. Moore mentioned the fact that some form of opiate had effect. Did any of them have atropin preoperatively? Atropin produces dryness of the throat and bronchial tree. If we give atropin there is cessation of activity on the part of the ciliated epithelia. We must not administer our anesthesia too deeply. Another very serious question should be asked, who operated on these patients? Go in any hospital, any afternoon, and you will find residents doing most

of the tonsillectomies. Too often the operation is not done under light anesthesia and the operation takes too long. We are really fortunate that more of these serious accidents do not occur.

Dr. Philip Stout: After the completion of the operation the patient is left to come out of the ether alone. With the few abscesses we see, we may see some in patients with tonsils unremoved. Some are secondary to sinus disease. One physician had sinus trouble and developed lung abscess without previous operation of any sort.

Dr. Gabriel Tucker: I saw about 12 of such cases last year. Infection occurs in the upper lobe because the patient is not in the upright position after operation and is in the recumbent position during anesthesia. Aspiration can thus occur in the upper as well as in the lower lobe.

Dr. James A. Babbitt: I had one experience with local anesthesia. The patient was a man, aged seventy years, and exhibited a rise in temperature after operation. A roentgenogram revealed collapse of an upper lobe. This collapse was due to mucus rather than the common affair. The child I report with pneumonia, had no symptomatology and the condition was not discovered until the crisis occurred. I agree with Dr. Ersner that tonsil operations should not be handed out promiscuously. A minute dose of atropin was given each of these patients before operation.

Present-day Theories Concerning the Etiology and Pathology of Nasal Sinus Infections

Dr. Benjamin D. Parish: Textbooks during the past years have devoted many pages to operative work and treatment, but have been somewhat indefinite on etiology and pathology. Lately, however, the journals have been replete with reports delving into the etiology and pathology of sinus infection. For purposes of discussion, Dr. Parish adopted Eggston's classification of sinus disease. The first type considered, hypertrophic or polypoid sinusitis, is characterized by thickened and edematous changes in the mucous membrane and periosteum and is usually associated with polypoid masses of the soft tissues and rarefaction and osteoporoses of the bone. The second type, that known as atrophic, fibrotic, sclerotic or arteriosclerotic sinusitis, is characterized by an increase in the fibrous connective tissue, stroma, thinning of the mucous membrane and metaplasia of the surface epithelium, associated with thickening of the periosteum and a condensing osteitis. The third type is a combination of the first two types in the same patient.

The histopathology of these types was minutely described and attention then called to the striking similarity of these findings to those observed in a study of cases definitely diagnosed as allergic.

The histopathology of allergic alterations was elaborated. In all of the work that has been done in the study of the regeneration of mucous membrane in these sinuses, all the investigators are struck by the fact that the regenerated epithelium is practically the same as the original tissue. A perfect pseudostratified ciliated columnar epithelium and complete regeneration of the epithelium and glands occur in from three to five months. Regeneration, while extremely persistent, is slightly retarded in the presence of active infection. The significant point is that much of the pathology that was present before operation, may very readily recur in the newly formed tissue. This is especially true in allergic cases.

It is impossible to pin the etiology or course of the disease upon the changes found. It is a question whether vascular changes are the primary ones, whether polyps are a primary cause or whether the thickened basement membrane or even the round-cell infiltration is where the pathology starts. For microscopic sections reliance must be had upon only advanced cases where the process has gone far enough to justify the drastic step of operation. More information concerning the early changes is sadly needed. Many factors may well be the inciting cause of sinus infection. It is a well-accepted fact that something more than pure bacterial infection is required in the etiology of sinus disease. General bodily debilitation and lowered resistance from prolonged exposure, mental overwork, loss of sleep, types of diet, endocrine imbalance and anatomic factors, both acquired and hereditary, play a distinct part in the etiology. An important factor in the sinusitis of childhood has been said to be a lack of vitamin A. Diets rich in vitamin A and with increased vitamin B content were found of benefit. Lack of vitamin A has been shown to lead to atrophy in the turbinates of a rat.

Mechanical irritation, as the accidental introduction of water into the sinuses in swimming and diving, has led frequently to outbreaks of sinus trouble. A rather important phase of the matter is the vasomotor factor in some of these infections, especially in the atrophic types; it has been felt that there was some pathology connected with the vasoconstrictors and vasodilators. The problem of allergy and allergic reaction in the nasal mucous membrane is one that deserves much attention and has much to support it. The mere fact that the pathology of the sinus is strikingly similar

in a series of allergic rhinitis cases and a series of cases picked at random is very significant. Possibly most of the cases of sinus disease have an allergic background. The persistent finding of eosinophils in the discharges and in the tissues is an argument for the allergic idea. It is still a question, however, which comes first, the allergy or the suppuration. Can we account for the fact that some cases showing other symptoms of allergy, asthma, eczema, etc., in addition to the nasal condition, are completely cured by treatment and clearing up of the ethmoids, while other cases may be operated upon, the ethmoids excised, etc., and yet neither the nasal symptoms nor the other allergic features subside? Dr. Parish feels that this may be accounted for by the fact that there are two types of suppurative ethmoiditis, that in which the allergic state is the background and the bacterial infection is merely secondary, and those cases in which the infection may be the primary one. Where infection is primary, radical treatment produces a cure. The greatest problem is to distinguish between the two types.

Discussion. Dr. Cutis C. Eves: I feel that a great many of these cases are caused by an unhygienic diet. Especially in children, I feel that diet and hygienic regulations are more important than our treatment. Modern life is responsible for much of this. The automobile, exposure to air, gas and tire dust and similar new conditions explain the increase in these cases. The modern craze for swimming is a factor, as the water washes off mucus and the mucous membranes are left unprotected. Swimming animals possess mechanisms whereby the mucous membranes are protected by special mechanisms. An individual tends to blow the nose more vigorously when water gets into the nose. Controlling the habits and diet of such patients is most important.

Dr. Philip Stout: I have had some experience with these allergic cases. Nearly every case of asthma has pathology of the sinuses. We find dark sinuses both by roentgenograms and by transillumination. Washing often removes little or nothing. Recurrence occurs a year or two after operation and upon reopening, the sinus is found as bad as before the first operation. It is possible that the etiological factor is a mould. Eosinophilia is said to be the opposite of suppuration.

Dr. James A. Babbitt: I am interested in one point. The ethmoidal sinuses were emphasized as a general point of infection in the sinus group. I have been impressed in the past by the large number of sphenoidal sinuses reported as normal. I wonder what Dr. Parish's experience has been?

Dr. Parish: I agree with Dr. Babbitt that the sphenoids are least often infected unless the patient has been referred by an ophthalmologist. The roentgenologist most often reports the ethmoidal sinuses as showing involvement.

Some Studies of Pathologic Temporal Bones in Children

Dr. Robert M. Wolff: The intention of the study was to supplement from a macroscopic standpoint, the excellent work on microscopic section of the ear both abroad, and more recently, in this country. An attempt was made to demonstrate pathology in the middle ear and mastoid, and frequent sequellæ of this pathology, viz., meningitis brain abscess, and lateral sinus thrombosis. A series of temporal bones was obtained in which there had been a definite otitis media or mastoiditis, followed by intracranial complications. While symptomatology, diagnosis and treatment were not included for purposes of brevity, an effort was made to explain the occurrence and possible pathways of infection of these complications. The cases were followed, autopsies attended, and temporal bones sectioned macroscopically to obtain the necessary data.

In order to fully appreciate what may happen in the course of a chronic or acute suppurative otitis media, a review of the infant and adult anatomy was given. The possible pathways of infection and result pathology were discussed in detail and the literature dealing with these reviewed.

To obtain the temporal bones intact, including a small portion of the external auditory canal, without causing external deformity, a portable motor with flexible shaft attached to a specially constructed circular saw was used. When a difficult angle was encountered, small drill holes were placed in line and the bone gently pried away from its attachments. The greatest possible precaution was taken to use the least amount of jarring in removing the bones. The specimens were immediately placed in Klotz solution No. 1 for twenty-four hours. They were then transferred to Klotz solution No. 2 in order to preserve as nearly as possible the original color of the mucous membrane and bone. After photographing the temporal bone, the specimens were frozen with dry ice and cut into sections approximately $\frac{1}{8}$ inch in thickness. Several specimens were cut almost parallel with the external auditory canal. The remainder were cut at right angles to it. This was accomplished by the use of the saw and motor mentioned before. The saw blade being quite thin and with small teeth, there was only a fractional amount of tissue lost in this method of cutting. Each section was examined carefully for evidence of bone necrosis, pus, adhesions, etc., and was

photographed in series, first, from without inward, then, the reverse side. Due to the thickness of these specimens, this double photographing was necessary since the small structures of the ear would otherwise have been missed. The sections were then mounted in flat glass jars, suspended so that both sides could be studied. The solution in the jars was again Klotz No. 2.

The first case, that of a male, aged thirteen months, was admitted to the hospital with a discharging right ear, a temperature of 103° and a leukocyte count of 22,000. A roentgenogram of the mastoid areas revealed cells of the semidiplotic type and the right side definitely hazy as compared with the left. Clinically, the child did not appear to have mastoiditis. Two days after admission the aural discharge decreased and the drum was incised with release of pus. On the following day the spinal fluid contained 80 cells per c.mm. but the fluid was sterile. A neurological consultant felt that a brain abscess or localized cerebritis had resulted from mastoid infection. The leukocyte count was now 32,000 with 74 per cent polymorphonuclear cells, and weakness was noted on the left side of the face, left arm and left leg. A simple mastoidectomy revealed fairly normal bone and no frank pus. Cultures from the mastoid were reported sterile. The temperature continued irregular and high and while the wound healed promptly, the ear continued to drain foul pus. Several blood transfusions and hypodermoclysis bolstered the general condition and for two weeks following operation the five spinal fluid specimens obtained were reported sterile and of normal cell content. A furuncle in the right frontal region was discovered and when opened drained a large amount of thick yellow pus. A roentgenogram showed absorption of bone in this area. A week later generalized convulsions developed, the spinal fluid became cloudy with 6800 white cells and 94 per cent polymorphonuclear cells and hemolytic streptococci were found in cultures. Antistreptococcus serum was given intraspinally, but the child died one week later.

At autopsy no macroscopic connection between the middle ear or the mastoid and the brain cavity could be found. The dura was firmly adherent over the entire right side. There was an osteomyelitic area in the right frontal bone. A large subdural abscess, containing several drams of pus, and enclosed by dense adhesions, was found in the right parieto-occipital area and in the posterior

part of the right frontal lobe, a small opening led to the osteomyelitic area in the frontal bone, the drainage route for the brain abscess. Definite meningitis extended over the entire left side of the brain.

This temporal bone was cut in a plane parallel to the posterior surface of the petrous portion. There was much pus found in the middle ear and the same was true of the labyrinth. No definite areas could be found showing how the pus travelled from the ear to the brain. However, with a purulent labyrinthitis, there were several pathways available for the infection to extend into the brain cavity. This pathway was not macroscopically evident. The labyrinthitis was probably an extension of the middle ear infection through the round window, since this showed some bony necrosis around the margin. The specimen also shows well marked tympanic cellulæ.

The second case, that of a female, aged nine years, had had a discharging right ear for one month. On admission, there was marked stiffness of the neck, over-active knee reflexes and a positive Kernig sign. The Babinski reaction was negative. Pus filled the right external canal and all drum landmarks were gone. The temperature was 103°. A simple mastoidectomy found extensive and necrotic mastoid cells. The lateral sinus was uncovered but the sinus wall appeared healthy. Beneath the tegmen antri and extending deeply were some cells that were necrotic. These were followed to a depth where it seemed advisable to stop due to the proximity of the superior semicircular canal. The spinal fluid from the first tap was cloudy and showed a cell count of 3000 increasing later to 13,000. A hemolytic streptococcus was cultured from the spinal fluid and a pure culture of B. influenzæ was obtained from the mastoid wound at operation. Eight days after admission the left arm and leg became flaccid. The mastoid wound showed no tendency to heal and drainage was profuse. On the day of death, the respirations dropped to 6 per minute but later rose to 20. The heart continued to beat for ten minutes after respirations ceased.

At autopsy, the brain was markedly edematous, causing a blockage of the cerebrospinal fluid and an increased intracranial pressure. The surface of the brain was covered with a thick, plastic exudate, typical of a purulent, generalized meningitis of streptococcic origin. The dura over the petrous bone appeared normal and intact, until it was stripped off when a dehiscence was revealed over the tegmen

antri as well as necrotic bone just external to the internal auditory meatus. Slight pressure over the dehiscence above the mastoid excavation caused pus to be forced up through the necrotic bone. There was no evidence of an extradural collection of pus or walling off process. When the right temporal bone was removed, including the lateral sinus groove, the specimen was cut at right angles to the external auditory canal. The mastoid roentgenogram had shown dipletic bone formation but on sectioning, there was evidence of pneumatic cell development of wide distribution. Cells were found above and below the bony wall of the internal ear as well as along the course of the eustachian tube. The bone in the extreme petrous tip showed some cell formation, perhaps accounting for the inward extension of infection rather than rupture through the external cortex. From the depths of the mastoid wound, the direct extension of the infection could be followed along the upper group of cells above the bony wall of the internal ear and just under the petrous cortex to become continuous with the dehiscence at the internal auditory meatus. B. influenzæ may have accounted for the bone necrosis found deep in the temporal bone as this organism has especial ability to attack bone. It is difficult to explain why, with a pure culture of influenzal organisms in the mastoid, a hemolytic streptococcic meningitis should develop. The B. influenzæ in this case may have been one of the secondary invaders of the mastoid and at the time of operation, was the predominating organism, for we must concede the fact that the meningitis had developed at least several days before admission and operation.

Discussion. Dr. George M. Coates: I want to compliment Dr. Wolff upon this splendid work. I want to ask him one thing. We have known for a long time that at birth the middle ear is filled with embryonal tissue. One of the cases shows the middle ear still filled with embryonal tissue at thirteen and a half months of age.

Dr. Philip Stout: I attend every autopsy I can. When an opening is made into the middle ear in infants and children, we find a jelly like substance. We even find it occasionally in adults. Whether it occurs just before death or not I do not know.

Dr. Wolff: It is difficult to explain the persistence of this material in the middle ear. In this case I assumed it was a case in which the embryonal tissue had not as yet disappeared.

NOVEMBER 16

Joint Meeting with the Section on Otolaryngology of the New York Academy of Medicine

Postoperative Pulmonary Complications; Are They Preventable? (Lantern Demonstration)

Dr. Chevalier Jackson: This subject may be considered best by presenting clinical cases arranged in three groups: (1) postoperative atelectasis; (2) post-tonsillectomic abscess; (3) cases of inspiration of instruments and other substances at the time of operation. These three groups are quite distinct. Postoperative atelectasis follows abdominal and occasionally pelvic operations; it is not primarily suppurative, and we do not see it after tonsillectomy or other operations about the head or upper air passages. In Group 2, on the contrary, a sudden onset with fulminating symptoms of abscess, including foul expectoration, follows imediately or shortly after a tonsillectomy or an operation about the upper air passages. This type of suppuration does not follow abdominal or pelvic operations unless due to septic infarct. In Group 3 there is the long symptomless interval characteristic of foreign body inspiration. There have been taken out in 10 cases portions of tonsil instruments, in 60 cases teeth dislodged at operation and in 1 case a tonsil. In all of these cases there was a symptomless interval, and no suppuration except in those cases in which the foreign body had been present in the lung for a long time, months rather than days. In the case of the tonsil, the tonsillar tissue, containing presumably all infections usually present in diseased lymphoid tissue, was in the bronchus seven months without producing abscess; for the first three months there were no symptoms. It is obvious that the whole question of postoperative suppuration is closely related to the whole problem of descending infections from the upper air passages to the lungs. Until we have determined the route of infection and the other etiologic factors, it will be impossible to answer the question as to whether or not these complications are preventable. The route by the air passages is very direct, as is proven by the foreign bodies lost down at operation; the route by the bloodvessels is equally direct, as shown by Fetterolf and Fox. The route by the lymph

channels may be from the field of operation in the upper air passages, or from the tracheobronchial tree by way of the local lymphatics in the lung, though many questions in regard to the latter group of lymphatics are as yet unanswered. There can be no doubt but that blood running down into the tracheobronchial tree is a thing that should be prevented so far as is possible. It not only is infected, but it furnishes a culture medium; it runs down into the tracheobronchial tree at practically every operation, and yet pulmonary abscess following tonsillectomy is exceedingly rare as compared to the number of tonsillectomies. One thing of importance in connection with the inspiration of blood is the fact that the cricopharyngeal pinchcock maintains the esophagus constantly closed except during swallowing. The larynx, on the contrary, is constantly open and it is located at the bottom of a funnel formed by the pharynx; into this funnel the blood pours from the wound in the tonsils and nasopharynx. It is true that some blood is swallowed; but the swallowing is intermittent, whereas the flow of blood into the larynx is constant.

Discussion. Dr. J. D. Kernan, of New York: Prevention of these complications is what the otorhinologist is doing. Prophylaxis involves removal of foci in the upper air passages, such as sinus disease, infected tonsils and teeth, etc. Would it not be possible to block the flow of blood into the trachea by giving anesthesia through the flag tracheal tube, and why not clean the lungs after operation by using intratracheal suction routinely?

Dr. Jackson: We need not tie ourselves to the question of blood going down the trachea. Various devices, such as Dr. Moore's tonsillectomy "Sump," have been devised to catch the blood and particles. The transmission of infection through the bloodvessels and lymphatics must also be taken into consideration.

Atresia of the Choanæ: Its Incidence, Types and Cause. (Lantern Demonstration)

Dr. J. Parsons Schaeffer: Atresia of the choanæ and the pharynx may be congenital or acquired partial or complete. Acquired atresias are more apt to be pharyngeal and congenital atresias choanal in location. The acquired atresias are due to a number of causes; syphilitic ulceration accounting for many of them; surgical trauma and other traumas appear to be the determining factors in some cases; then follow, in order, diphtheria, tuberculosis and other

simple inflammations. Many of the acquired atresias are, however, not located at the position of the choanæ, but lower in the pharynx, really resulting in an occlusion between the nasal pharynx and the oral pharynx.

This paper limits itself to a consideration of congenital atresia of the true choanæ. The occlusion may be absolute or partial; the atresic mass epithelial, membranes, osseous or combinations of these. While the condition appears relatively uncommon, its occurrence is doubtless more frequent than is generally believed. In all likelihood some atresias of the posterior nares pass unobserved in the stillborn and in infants that die shortly after birth.

In the living infant with absolute occlusion of the choanæ, early signs suggestive of nasal obstruction are manifested. When the occlusion is partial, even though the choanal apertures are small, the real condition may not be recognized for some time. Indeed, some individuals go into adult life with partial atresia of the choanæ, variously hindering the intake of air by way of the nose. Spontaneous rupture of the atresic mass, especially if thin and membranous, may occur early in infancy. As a rule, operative measures are, however, required to relieve the obstruction.

The explanation of congenital atresia of the true definitive choanæ or posterior nares must be sought primarily (1) in the nasal fossæ and the early mouth cavity, and (2) the behavior of the floor of the early nasal fossæ dorsal to the primitive choanæ.

Should the primitive choanæ just dorsal to the primitive palate not form during the process of development, the epithelial lining of the primitive nasal fossæ and the epithelial lining of the primitive oral cavity would remain intact, each separated from the other by the thinned-out mesenchymal floor of the early nasal fossæ. The continued dorsal growth of the nasal fossæ into the mesenchymal mass of the nasofrontal process would ultimately, in cases of maximal growth, lead to a condition in which the epithelium of the nasal fossæ would come into contact with the epithelium of the foregut, *i. e.*, the nasal portion of the pharynx, with a variable amount of mesenchymal tissue between the epithelial surfaces. This would result in blindly-ending, definitive nasal fossæ dorsally; in other words, an atresia of the choanæ of posterior nares. If the amount of mesenchyme between the pharyngeal and nasal epithelial should remain thick, an osseous atresia would ultimately be formed. The atresia of the choanæ would, therefore, be membranous, osseous

or a combination of these, depending on the degree of dorsal growth of the nasal fossæ and the extent of the resorption of the mesenchyme between the nasal and the pharyngeal epithelia.

Even though the primitive choanæ are established normally in the roof of the mouth, should the floor of the primitive nasal fossæ dorsal to the primitive choanæ not be resorbed, there could be no connection with the nasopharynx save by way of the mouth and the oral pharynx.

The palatal processes developing beneath the floor of the early nasal fossæ, and fusing with each other in the midline in the formation of the secondary palate, would come into intimate relationship with the persistent primitive floor of the nasal fossæ dorsal to the primitive choanæ and with the nasal floor when the primitive choanæ are not established. Fusion between the palatal processes and the primitive nasal floor would in time take place.

Discussion. Dr. E. Ross Faulkner, of New York: I believe it is possible that a large number of babies and children dying from respiratory difficulty may have some similar congenital condition. The patency of the choanæ should be determined before operating rather than suppose that adenoids are responsible for obstruction.

Discussion of Some of the Recent Developments in Regard to Otosclerosis, Cholesteatoma and the Static Labyrinth

Dr. George M. Coates: As the title suggests, this is a résumé of certain phases of work of several distinguished investigators; work that should be of interest to every otologist.

Otosclerosis

This chapter is based upon the work of Polvogt and Guild in Baltimore. The usual pathology of otosclerosis accepted by otologists is confined to the area surrounding the oval window, causing ossification of the annular ligament with fixation of the footplate of the stapes in the oval window. Polvogt and Guild had the opportunity of studying, by serial section, many temporal bones of patients of whom audiometric records had been made. In some of these they discovered definite areas of otosclerosis which had not caused the usually accepted symptoms and signs and in some were not found in the usual locations. Guild reported 8 cases, and Polvogt

added 2 more, where the history, familial and personal as well as clinical symptoms, did not suggest otosclerosis, but where definite otosclerotic areas were found on sectioning. Some of these were at a very early stage, some active and others entirely quiescent, although evidently of long duration, partially confirming the clinical fact that the disease is at times self-limited and is not uniformly progressive.

Because of the lack of clinical symptoms, many cases were classed as "early" although they may have been old pathologically. The changes may begin as early as the first year of life and do not always first appear near the anterior margin of the oval window, nor is the fissure ante fenestram always involved.

In early otosclerosis about the round window, when it reaches the stage of ankylosis, we can usually diagnose the uncomplicated cases. In otosclerosis of other regions, affecting the cochlea and producing nerve-type deafness, Nager, of Zurich, showed that such cases could not be distinguished clinically from those produced by other causes.

Cholesteatoma

Based on Wittmaac's theories of pneumatization of the temporal bone and some practical applications worked out in Kopetzky's clinic, Almour advocates the utilization of the cholesteatomatous matrix for the epidermatization of the radical mastoid cavity. The normally pneumatized mastoid process, it is now believed, is formed by invasion and replacement of the bone marrow by embryonal myxomatous connective tissue, which has filled the middle ear and antrum at birth. This tissue with its limiting epithelium is drawn into the intertrabecular spaces and by contraction eventually lines them, thus affording a more or less direct communication with the antrum and middle ear. Any interference with this normal development produces either a diploic or sclerotic mastoid—in other words, an undeveloped mastoid process, or possibly a mixed type, depending upon the stage of development when the interference occurred. Interference may be by an uninfected foreign body, such as meconium or vomitus, or by microbic infection taking place before development. In either instance a fibrosis is induced which interferes with the ingrowth of the subepithelial tissue into the mastoid process. Thus a sclerotic mastoid process is not viewed as pathological in itself or the result of a chronic infection in the middle ear or antrum.

Where it can be proved by *x*-ray that the mastoid process is nonpneumatic and wholly sclerotic, Kopetzky and Almour confine their radical mastoid operation to the removal of the thick cortex overlying the antrum, without any attempt to excavate the remainder of the process which is considered healthy bone. The cholesteatomatous mass is removed with care from the uncovered antrum, the posterior canal wall reduced and the middle-ear cavity treated in the usual orthodox manner. This procedure materially shortens the time of operation and leaves a much smaller area to be epidermatized. It is considered very necessary, in making the plastic flap, that the meatal opening be ample, in order to provide adequate escape for epithelial débris, as otherwise cholesteatoma may redevelop. In other words, cholesteatoma is an attempt at a reparative process, which may succeed perfectly if the avenue of escape for exfoliated epithelial débris is adequate. Therefore, these observers advocate the retention of the cholesteatomatous matrix, which is nothing more than an epidermal covering similar to the one which the operator hopes will eventually line his radical cavity. We now believe that the old teaching, following the observation of Kirchner, is unnecessary, the cause of recurrence of the cholesteatomatous mass being an inadequate outlet.

Studies of the Equilibrial Mechanism of the Frog

McNally and Tait have reached definite conclusions upon the function of certain portions of the static labyrinth, at least in the frog, which seem very logical and clarifying. They believe the saccule (with its macule and otoliths) has no function in the production of equilibration, but appears to be a rudimentary part of the auditory system.

Response to angular acceleration in rotation (slow speed) is a function of the semicircular canals, whereas response to centrifugal force (rapid turning) is otolith stimulation in the utricle. The response to gravity gained by tilting the table is similar to the above, as it is abolished by cutting the nerve to the utricle.

Static function arises from the semicircular canals, while the otoliths serve as a kinetic mechanism. If both labyrinths are removed from a frog, it may assume normal or unusual positions, but it maintains its position in spite of the lack of labyrinths and, therefore, the position is due to a neuromuscular mechanism.

When a normal frog is placed upon a plane and the plane is tipped

slowly so that the movement is below the threshold of stimulation of the canals, the frog responds by discontinuous shifts. If the utricle were steadily stimulated the shift would be steady and not discontinuous.

If we now remove one labyrinth from a frog, the animal leans to one side. By manipulating such a frog and bringing its posture to the midline, the frog maintains its new symmetrical position when released; the moment the head is jarred or moved, however, the frog regains the asymmetrical position. A decerebrated frog is normal in labyrinthine movements, but voluntary movements are curtailed. It is the necessary jar of the utricular otolith that causes response. There is no steady pressure, but there is a sudden stimulus.

By removing all six canals in a frog, only the utricles remain as active vestibular end-organs. The posture is normal at rest, but any spontaneous movement causes the animal to sway and the jumps are not straight. The canal response is so rapid that response occurs in 1/25,000 of a second.

In the frog, maintenance of position is evidently a muscular affair. When the canals are present, they act to keep the head level and prevent the sway which the utricles would otherwise bring about. The static mechanism is evidently muscular.

Discussion. Dr. Samuel J. Kopetzky, of New York: Since the compact bone in the mastoid should not be thought of as pathologic, and since egress only is necessary to stop cholesteatoma, why do more than remove the diseased portion and afford ample drainage? There is one exception to this: that is, when the cholesteatomatous matrix involves intracranial structures; then, of course, the whole matrix should be removed. In otosclerosis the lesion is in unique bone. The lesion is not unique for it is found in other diseases, such as cretinism, osteomalacia, von Recklinghousen's disease, etc., There is a functional disturbance in the nervous system. Three important factors are: (1) a "trip" in the calcium fixation somewhere in the body. (2) The bony labyrinthine capsule is the only bone developed fully at birth. Therefore, the time to study it is in the intrauterine period. If it were possible to get fetuses from families with an heredity to otosclerosis, we might find the cause. (3) Deafness is only secondary to nerve degeneration after birth.

The Anatomy of the Tympanic Membrane (Demonstration with Manikin and Preparations)

Dr. O. V. Batson (by invitation): Since the clinical approach to the membrana tympani is by way of the external auditory canal,

the changes that occur in the canal with age are here of interest. In infancy the canal is arched upward in its middle portion. As age advances this curve commonly becomes flattened, so that by middle life the course of the canal approaches the horizontal. This seems to be associated with the flattening of the middle cranial fossa. Casts of the canal made at various ages show that the manipulations used to facilitate examination of the drum membrane are only useful in aligning the auricle with the canal and probably produce no change in the canal itself.

Numerous ear microscopes have been developed to make clinical observations. The clumsy and complicated ones defeat their purpose. It should be remembered that the resolving power of the objective and not the magnification of the ocular is significant in a diagnostic instrument. It is then readily seen that the small telescopic magnifiers give as much wealth of detail as the larger instruments now available.

Specimens prepared for this study agree with the published accounts of the vascular supply of the drum membrane. The vessels radiate from the malleus handle to the margin. The dual connections—the central vessels and the peripheral plexus—indicate in part the reason for the ready regeneration of the membrana following pathological or surgical puncture.

The fibrous structure of the membrana itself was investigated by the method discovered by Dupuytren and elaborated by Langer. A pointed round instrument thrust into the skin produces a slit. The direction of the slit depends upon the direction of the predominating fibers. This phenomenon was found to hold for the drum membrane, which was for the first time examined by this method. The method is important because it shows the direction of the major portion of the fibers, the slits are larger in the direction of the major fibers. It is known, further, that rapid natural closure follows any skin incision with the elevages, while drainage is secured by making the incision across the direction of the clefts. There are two groups of clefts produced in the drum membrane, circular and radial. The circular clefts are small and fall into a central and a marginal group. The radial clefts are coarse slits, they are at right angles to the handle of the malleus and radiate from its tip. Examination of the membrane by polarized light contributed no additional information.

This study indicates that the usual paracentesis tympani, between the malleus and the posterior margin, separates the membrana so

that there is an adequate blood supply on either side of the cut and, further, the incision is so placed in relation to the fibers that maximum drainage will be obtained.

Discussion. Dr. Marvin F. Jones, of New York.

DECEMBER 21

Preventriculosis (So-called Cardiospasm). Diagnosis and Treatment. (Moving Picture Film Demonstration)

Dr. Chevalier L. Jackson: The film showed briefly the physiology of swallowing, and then the pathological physiology of "preventriculosis." "Preventriculosis" is the term preferred by Chevalier Jackson to designate the condition commonly known as "cardiospasm." This condition is one whose etiology and pathogenesis have not as yet been entirely determined, but it can at least be said that in typical cases of this disease there occurs a functional stenosis at the level of the diaphragm, associated with more or less dilatation of the esophagus above. The "diaphragmatic pinchcock," probably composed chiefly of the crura of the diaphragm, *fails to open* as it normally should in the deglutitory cycle, though on passage of an esophagoscope or other instrument, no more than the usual resistance is met with at this point.

The typical roentgen-ray appearances of "preventriculosis" were then shown. While roentgen-ray examination is of great diagnostic aid, the diagnosis should always be confirmed by subsequent esophagoscopy to rule out an organic lesion. The technic of diagnostic esophagoscopy was also demonstrated in the film.

The most satisfactory instrument for therapeutic dilatation is, in our opinion, a pneumatic dilator of the type used by Mosher, with opaque lines in the rubber bag which permit one to place the instrument accurately under fluoroscopic guidance, as well as to observe the outline of the indentation produced on the bag, and the amplitude of dilatation obtained. This apparatus, and the technic of its use, were shown in the film. The rubber tubing is calibrated in order to facilitate the taking of measurements at the first (fluoroscopic) dilatation, for subsequent use when fluoroscopic guidance is not used, and this feature was also demonstrated.

Laryngofissure for Early Laryngeal Cancer. (Moving Picture Film Demonstration)

Dr. Gabriel Tucker: The treatment of cancer of the larynx by laryngofissure necessitates early diagnosis. The film outlines the steps for early diagnosis, general examination, *x*-ray examination, repeated mirror observations and study. The diagnosis of cancer should be confirmed in every case by biopsy before either treatment by surgery or irradiation is planned. A demonstration of the direct laryngoscopic technic for biopsy was shown. Early cancer of the larynx originates most frequently in the anterior portion of the glottis and is amenable to surgical removal by laryngofissure. Laryngofissure is applicable only to this type of growth. The more extensive lesion and lesions in other locations may be treated by a thyrotomy incision with wider removal of tissues. These operations, however, should be termed *partial laryngectomy*. The term laryngofissure should be restricted to the removal of the tissue within the thyroid cartilage only. The technic of Chevalier Jackson for growth on the cord, and in the anterior commissure was illustrated by drawings. Also the technic of Sir St. Clair Thomson. It was suggested that the Sir St. Clair Thomson technic may be used also, for partial laryngectomy because of the wide exposure that it gives inside the larynx. Local anesthesia only is required for a laryngofissure. In a series of 50 cases of laryngofissure and partial laryngectomy there was no operative mortality. Management of complications following laryngofissure was discussed. The method of treatment of wound infection and the direct laryngoscopic removal of excessive granulations following laryngofissure was demonstrated. The treatment of recurrences which occur in less than 15 per cent of the cases of laryngofissure was demonstrated in a patient who had a laryngofissure, partial laryngectomy and total laryngectomy, there being no recurrence after the total laryngectomy over a period of one and a half years. The following conclusions were offered:

1. Chronic hoarseness is a constant symptom in intrinsic cancer of the larynx.

2. The choice of laryngofissure for surgical cure in intrinsic cancer of the larynx should depend on the location and extent of the lesion. The cell characteristics are of secondary importance.

3. Laryngofissure for early intrinsic cancer of the larynx will cure

over 80 per cent of the cases, saving the patient's larynx as well as his life.

4. A more radical operation can still be done in cases of recurrences following laryngofissure if the patient has the proper follow-up observation.

Operation on Abscess of the Temporosphenoidal Lobe. (Moving Picture Film Demonstration)

Dr. Walter Roberts and Dr. Henry Dintenfass:

Discussion. Dr. Henry Dintenfass: This series of operations was successfully performed by Dr. Roberts whose excellent judgment in the diagnosis and in the timing of the therapeutic procedures brought about the complete recovery of the patient.

At the first operation, when the simple mastoidectomy was performed, and the cofferdam placed into position, I did not have my camera with me, so we have substituted a drawing in the film to show what occurred.

The particularly interesting features in this case, of course, were the diagnosis and method of treatment. In the symptomatology it will be observed that the slow pulse, photophobia, headache, and clouded mentality were merely the manifestations of increased intracranial pressure as are associated with brain abscess in any location. The presence of aphasia, however, was a localizing symptom and indicated definitely that the lesion was temporosphenoidal. Actually, the superior temporal convolution, or Heschel's convolution, was affected. In this connection it should be mentioned that the aphasia was sensory in type, since the patient could understand and recognize objects but he was unable to name them; yet he could repeat the name of an object after he had heard it. This is to be differentiated from motor aphasia, where objects cannot be named at all. The lesion for motor aphasia is in Broca's region, the inferior frontal convolution. It was fortunate that the abscess was not in the so-called "silent" area, that is, on the right side of the brain in a right-handed individual, otherwise, the localizing symptom of aphasia would not have been present.

As has been mentioned, papilledema was absent. Neither did vomiting occur, notwithstanding the size of the abscess. According to Kopetzky, these are late symptoms of abscesses in the temporosphenoidal region. In cerebellar abscess papilledema is apt to exist earlier in the course of the disease because of the proximity of the pus pocket to the foramina of the fourth ventricle, with the consequent blocking of the ventricle by pressure.

The use of the cofferdam to seal off the inflammatory process and to prevent meningitis was a lifesaving procedure. In incising the dura great care was taken not to injure the middle meningeal artery. The incision in the dura was made parallel to the temporal convolutions.

The problem of drainage, one of most importance in brain abscesses, was taken care of by the rubber tube, the abscess being completely emptied

after the first insertion. This would give the impression that some form of abscess capsule existed, notwithstanding the acute type of lesion.

It is also interesting to note that the aphasia cleared up the next day following the drainage, but the slow pulse continued for some days after the operation, which demonstrates that the adjustment to normal of the increased cerebral pressure is a slow procedure.

Dr. Roberts is to be congratulated on his excellent management of the case.

Total Laryngectomy. One-stage Operation. (Lantern Demonstration of the Various Phases and Steps in this Operation)

Dr. Fielding O. Lewis: Lantern slide demonstration of the various phases and steps in the one-stage operation in total laryngectomy.

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

PAPERS READ DURING 1932

JANUARY 25

Dr. Burgess Gordon, Dr. O. S. Kough (by invitation) and A. Proskouriakoff, Ch. E. (by invitation), "The Immediate Effects of Calcium Levulinate Administration."

Dr. John H. Arnett and Dr. LeRoy M. Ennis, D.D.S. (by invitation), "The Teeth as Foci of Infection. Results of Routine Dental *X*-rays in College Students."

Dr. David W. Kramer (by invitation) and Miss Freda Ward (by invitation), "A Study of Glucose Tolerance Curves in Tuberculosis: Observations on One Hundred Cases."

Dr. J. W. Cutler (by invitation), "The Blood Sedimentation Test in the Management of the Pneumothorax Patient."

Dr. F. M. McPhedran, "Tuberculous Activity as Reflected in Signs, Symptoms, the White Count and the *X*-ray Film."

FEBRUARY 22

Dr. Joseph Edeiken (by invitation) and Dr. Charles C. Wolferth, "The Clinical Significance of the Deep Q-wave in Lead III of the Electrocardiogram."

Dr. Thomas A. Shallow, "The Influence of the Parathyroid on Bone Pathology."

Guest Speaker: Dr. Arthur M. Fishberg, New York City, "Hypertensive Encephalopathy."

MARCH 28

Dr. Isolde T. Zeckwer (by invitation) and Dr. Edward Rose, "A Malignant Tumor of the Thyroid with Metastasis Solely to the Kidneys; Death from Renal Insufficiency."

Dr. Gordon J. Saxon, "Indicanuria."

Dr. Edward Weiss, "Calcified Plaque of the Aorta at the Entrance of a Patent Ductus Arteriosus. A Point in Diagnosis."

Dr. J. Alex Clarke, Jr. and Miss Gertrude Holmes (by invitation), "Comparative Eosinophilia of Blood and Sputum."

Dr. F. William Sunderman, "The Concentration of Electrolytes and Non-electrolytes in the Serum following Insulin Administration in Diabetic Patients."

APRIL 25

Dr. John T. Farrell, Jr., "Changes in the Roentgenographic Appearances of the Chest Secondary to Rib Resection."

Dr. Mitchell Bernstein (by invitation), "Study of Syphilis as it Appears in a Medical Dispensary."

Dr. Harry P. Schenck and Dr. Richard A. Kern, "Clinical Experiences with Synthetic Ephedrin."

Dr. M. G. Wohl (by invitation), "Masked Hyperthyroidism."

Miss Edith M. Hall (by invitation) and Dr. Stanley P. Reimann, "A Chick Heart Method of Biological Assay of Digitalis."

MAY 23

Dr. Alfred Gordon, "Motor Paralysis of Individual Nerve-trunks Following Administration of Prophylactic Serum."

Dr. Frank W. Burge (by invitation), "Intestinal Insufflation with Air in Idiopathic Chronic Diarrhea. Preliminary Clinical Observations."

Dr. Alexander Uffe (by invitation), "Urticaria."

Dr. John Brennan (by invitation) and Dr. Russell S. Boles, "A Case of Tularemia."

Dr. Alexander Margolies (by invitation), "Factors Influencing the First Heart Sounds."

OCTOBER 24

Dr. Lewis H. Hitzrot (by invitation), "Pure Lipoid Nephrosis with Report of a Case."

Dr. V. M. Moon, "A Combination of Factors in the Etiology of Cirrhosis."

Dr. Joseph Edeiken (by invitation), "The Heart in Spinal Deformities."

Dr. Jos. C. Doane, "Diseases of the End Arteries."

NOVEMBER 28

Dr. Harold Goldburgh (by invitation), Dr. David Goltman (by invitation) and Dr. Walter Crocker (by invitation), "Liver Extract for Intramuscular Use: Its Advantages in the Treatment of Pernicious Anemia."

Dr. Harold W. Jones and Dr. Leandro Tocantins (by invitation), "Blood Transfusion *versus* Iron in the Treatment of Secondary Anemia; A Study in Hospital Economies."

Dr. H. K. Mohler and Dr. B. L. Crawford, "Pathological Findings in Hearts with Auricular Fibrillation."

Dr. Joseph V. Klauder and Herman Brown (by invitation), "Cystine in its Relation to the Growth of Hair; Report of Laboratory and Clinical Studies."

DECEMBER 19

Dr. Carl J. Bucher and Dr. Jo Ono (by invitation), "Tracheoesophageal Fistula of Syphilitic Origin; with Report of a Case."

Dr. Alexander Margolies (by invitation), "The Classification of Gallop Rhythm and the Physiological Third Heart Sound."

Dr. B. L. Crawford and Dr. A. Cantarow, "Fatal Arsphenamin Reaction, with Unusual Pathological Lesions."

Dr. John A. Kolmer, "Oral Immunization Against Experimental Pneumococcus Meningitis, Pneumonia and Septicemia."

PROCEEDINGS

OF THE

SECTION ON PUBLIC HEALTH AND INDUSTRIAL MEDICINE

DECEMBER 9

PAPERS READ DURING 1932

Dr. Leroy U. Gardner, Director, The Saranac Laboratory for the Study of Tuberculosis of the Edward L. Trudeau Foundation (by invitation), "The Pathology of Certain Forms of Pneumonokoniosis."

Mr. J. J. Bloomfield, Sanitary Engineer, United States Public Health Service (by invitation), "The Determination and Mitigation of Industrial Dust."

Dr. A. J. Lanza, Assistant Medical Director, Metropolitan Life Insurance Company, New York (by invitation), "The Control of Silicosis."

Dr. Henry K. Pancoast, The University Hospital, Philadelphia, Pa. (by invitation), "The Explanation of the Pathology of Silicosis by Roentgenological Findings."

Dr. Eugene P. Pendergrass, The University Hospital, Philadelphia, Pa. (by invitation), "The Diagnosis of Silicosis by Roentgen Examination."

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

PAPERS READ DURING 1932

JANUARY 12

Dr. Harold W. Jones and Dr. Leandro Tocantins (by invitation), "The History of Purpura Hæmorrhagica."

Dr. James Hendrie Lloyd, "Dr. Guillotin's Idea."

Dr. Herbert Fox, "Kitasato."

Dr. Leopold Vaccaro (by invitation), "Dante's Relationship to Medicine."

FEBRUARY 8

Commemorating the Bicentenary of Washington's Birth.

Dr. Francis R. Packard, "Washington and the Medical Department of the Continental Army."

Dr. Wyndham B. Blanton, Richmond, Va. (by invitation), "Washington's Medical Knowledge and its Sources."

Dr. Fielding O. Lewis, "Washington's Last Illness."

Demonstration of souvenirs by Dr. David Riesman and Dr. Walter G. Elmer.

MARCH 14

In coöperation with the Henry Phipps Institute, the Philadelphia Health Council and Tuberculosis Committee, and the Association of Tuberculosis Clinics commemorating the 50th Anniversary of the Announcement by Robert Koch on March 24, 1882, of the discovery of the tubercle bacillus.

Dr. Lawrence F. Flick, presiding.

Dr. H. R. M. Landis, "Reception of Koch's Announcement in the United States."

Dr. Theobald Smith, Princeton, N. J. (by invitation), "Koch's Views on the Stability of Species among Bacteria."

Dr. Lawrason Brown, Saranac Lake, New York (by invitation), "What Koch Meant to Tuberculosis."

Dr. Damaso de Rivas, "Personal Reminiscences of Robert Koch."

APRIL 11

Commemorating the Centenary of the Death of Johann Wolfgang von Goethe.

Dr. Edward A. Schumann, "Goethe as a Naturalist."

Dr. Burton Chance, "Goethe and his Theory of Colors."

Dr. Charles W. Burr, "Nostradamus, Astrologer and Physician."

Dr. J. Gershon-Cohen and Edward H. Landis, Ph.D. (by invitation), "Roentgen, Phases of his Life and a Practical Demonstration of the Physical Apparatus used by him in the Discovery of the Roentgen Ray."

OCTOBER 10

Dr. James M. Anders, "Hiram Corson, M.D., Clinician, Pioneer and Reformer."

Dr. Fielding H. Garrison, Colonel, U. S. A., Baltimore, Md., "The Children of Robert and Clare Schumann—an Introduction to Familienforschung."

Dr. C. N. B. Camac, New York City (by invitation), "The Greek Emblem of Medicine."

Dr. John B. Becker, "A Pioneer Proctologist, being a Review of John Arderne's Treatises of Fistula-in-ano, Hemorrhoids and Clysters."

NOVEMBER 14

Sixth Annual Students' Night.

History of Physical Diagnosis.

Dr. Robert W. Buck, Boston, Mass. (by invitation), "Physical Diagnosis prior to Auenbrugger."

Dr. Thomas McCrae, "Auenbrugger and Laennec and their Contributions to Physical Diagnosis."

Dr. Thomas Barnes Futcher, Baltimore, Md. (by invitation), "Physical Diagnosis between Laennec and Roentgen."

Dr. William Egbert Robertson, "Later Additions to Physical Diagnosis."

Exhibition of instruments and books relative to physical diagnosis through the courtesy of Dr. William Egbert, Robertson, Dr. Harold Robertson and Messrs. George E. Pilling Company.

DECEMBER 12

Commemorating the Bicentenary of the building of the Philadelphia Almshouse from which the Philadelphia General Hospital developed.

Dr. Joseph McFarland, "The History of Nursing at Blockley."

Dr. William S. Middleton, Madison, Wis. (by invitation), "Clinical Teaching in the Philadelphia Almshouse and Hospital."

Dr. Edward B. Krumbhaar, "The Study of Pathology at Blockley."

Dr. D. J. McCarthy, "Fifty Years of Medicine in Blockley."

Dr. Harold D. Palmer, "de Warville's Description of the Almshouse in 1788."

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1932

Mr. President: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1932:

Number of volumes in the Library	169,045
Number of unbound "Reports" and "Transactions" (included in above)	24,620
Number of unbound "Theses" and "Dissertations" .	24,523
Number of unbound Pamphlets	205,743
Manuscripts	445

Included in the above are 4467 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2972 volumes more or less incomplete.

The duplicates which number 9240 volumes at this date are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	124,384	2,972	127,356
Lewis Library[1]	13,855	44	13,899
On permanent deposit:			
S. D. Gross Library . .	3782	4	3786
Library of the Obstetrical Society of Philadelphia	217	0	217
Mütter Museum[2] . . .	190	0	190
			145,448

Received during the year from all sources 3611 volumes, 13,110 pamphlets and 14,950 numbers of various periodicals.

[1] The books of the Lewis Library are in process of being merged with the General Library.

[2] The books of the Mütter Museum are not permitted to be removed from the College building.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	3,611	13,110	14,950
Lewis Library	0	0	0
S. D. Gross Library	1	0	0
By purchase, General Account	607	0	0
In exchange	344	9	103
	4,563	13,119	15,053

Accessions (including 4 volumes of "Reserves"), 2339.

	Volumes.
General Library	2,338
Lewis Library	0
S. D. Gross Library	1
	2,339

Total increase in the number of volumes for the year, 2339.

Photographs received in response to requests sent out during year, and voluntary gifts: Fellows of the College, 1; other than Fellows, 0.

Total number of portraits listed, 23,941.

The individual "Donors" for the past year ending, November 1, 1932, number 400. This represents 1261 distinct presentations. Each gift is duly acknowledged.

The following list shows individual gifts of twenty volumes and over; also the number of volumes presented by the various publishing houses:

	Volumes.
Dr. J. H. Austin	63
Dr. J. M. Deaver	63
Dr. F. X. Dercum	591
Dr. R. S. Hooker	379
Dr. E. B. Krumbhaar	20
Dr. C. M. Montgomery	129
Pennsylvania Hospital	80
Dr. D. B. Pfeiffer	20
Dr. Joseph Walsh	68
Dr. Richard Wilson	115
Univ. of Penna., Dept. of Pharmacology (Dr. A. N. Richards)	30

From the publishing houses of:

	Volumes.
Baillière, Tindall & Cox	2
P. Blakiston's Son & Co.	12
F. A. Davis Company	10
Lea & Febiger	16
J. B. Lippincott Company	14
C. V. Mosby Company	2
W. B. Saunders Company	50

The Library is indebted for gifts of pamphlets and unbound periodicals to the following donors:

Dr. H. B. Allyn
Dr. J. H. Arnett
Dr. A. P. C. Ashhurst
Dr. J. H. Austin
Dr. E. J. G. Beardsley
P. Blakiston's Son & Co.
Dr. C. W. Burr
Dr. Burton Chance
Dr. S. S. Cohen
Mrs. F. X. Dercum
Dr. W. A. N. Dorland
Dr. G. M. Dorrance
Dr. A. A. Eshner
Mr. C. P. Fisher
Col. F. H. Garrison
Dr. B. C. Hirst
Mr. P. B. Hoeber
Dr. W. H. Hoffmann
Dr. H. H. Jenks
Dr. J. H. Jopson
Dr. W. W. Keen
Dr. E. B. Krumbhaar
Dr. F. H. Leavitt
Dr. Joseph Leidy
J. B. Lippincott Company
Dr. J. W. Maliniak
Dr. Edward Martin
Milbank Memorial Fund
Dr. C. M. Montgomery
Dr. E. J. Morris
Dr. T. R. Neilson
Dr. C. A. O'Reilly
Dr. F. R. Packard
Pennsylvania Hospital
Philadelphia Pathological Society
Dr. David Riesman
W. B. Saunders Company
Dr. G. E. deSchweinitz
Dr. W. J. Taylor
U. S. Department of Agriculture
U. S. Department of Commerce
U. S. Department of Interior
U. S. Public Health Service
Dr. Joseph Walsh
Dr. P. F. Williams
Dr. J. C. Wilson
Dr. Richard Wilson
Dr. C. S. Witherstine
Dr. A. C. Wood
Dr. William Zentmayer

589 new publications were added to the Library during the past year, viz: United States, 270; Great Britain and Colonies, 83; France, 79; Germany, 144; Italy, 5; all other countries, 8. Of these new publications, 11 were written by Fellows of the College.

22 volumes were presented by the following authors or editors:

Dr. K. E. Appel	Dr. John Ruhräh
Dr. E. D. Bond	Dr. J. P. Schaeffer
Dr. R. H. Ivy	Dr. J. F. Schamberg
Dr. W. F. Koch	Dr. J. M. Taylor
Dr. E. B. Krumbhaar	Dr. G. T. Vaughan
Dr. B. B. V. Lyon	Dr. H. R. Viets
Dr. J. F. Montague	Dr. J. F. Williams
Dr. F. R. Packard	Dr. P. F. Williams
Dr. W. A. Pusey	Dr. W. H. Wilmer
Dr. A. Randall	Dr. C. S. Wright

16 volumes were sent by the publishers at the request of the following authors or editors:

Dr. S. H. Brown	Dr. O. H. Petty
Dr. Abraham Cantarow	Dr. W. C. Posey
Dr. H. W. Cattell	Dr. O. Rahn
Dr. A. Granger	Dr. J. W. Sluss
Dr. H. A. Hare	Dr. E. Thornton
Dr. L. J. Menville	Dr. M. Trumper
Dr. W. H. Meyer	Dr. J. F. Williams

Cleveland Academy of Medicine

Summary of the "Funds:"

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	27	$143.37
Luther S. Bent Fund	7	10.46
William T. Carter Fund	14	25.27
Gerardus Clarkson Fund	1	1.35
Francis X. Dercum Fund	26	208.72
Louis A. Duhring Fund	20	169.87
Augustus A. Eshner Fund	44	244.49
John D. Griscom Fund	36	106.78
Richard H. Harte Fund	45	388.63
Charles T. Hunter Fund	2	24.57
William F. Jenks Fund	39	297.76
Oliver A. Judson Fund	9	70.98
William V. and John M. Keating Fund	5	47.18
William W. Keen Fund	36	212.63
Library Endowment Fund	141	625.29
Horace Magee Memorial Fund	124	414.49
J. Ewing Mears Fund	29	300.39
Charles K. Mills Fund	1	2.35
Weir Mitchell Fund	50	275.03
Carried forward	656	$3,569.61

	Volumes purchased.	Cost.
Brought forward	656	$3,569.61
E. E. Montgomery Fund	17	111.60
John H. Musser Fund	4	42.55
Elizabeth K. Newcomet Fund	6	21.22
William F. Norris Fund	19	127.56
Charles A. Oliver Fund	8	54.97
Philadelphia Medical Society Fund	3	6.79
Lewis Rodman Fund	14	86.62
Douglas Stockton Warren Fund	24	112.66
John F. Weightman Fund	3	10.25
J. William White Fund	15	181.99
Caspar Wistar Fund	16	123.01
S. Lewis Ziegler Fund	21	59.83
	806	$4,508.66
From the General Account—by purchase	607	$4,250.66

Special accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	85	$457.15
Fund for rare and valuable books	19	429.20
S. D. Gross Library Account	0	0

Catalogue Endowment Fund—on account of salaries	$46.50
Louis A. Duhring Fund—for general purposes of the Library	12,250.00
Morris Longstreth Fund—on account of salaries	1,098.00
George B. Wood Fund—Library supplies, stationery, etc.	359.84
Henry Leffman Trust Fund—miscellaneous expenses in aid of the Library	237.20

Number of volumes bound, 1795.

	Visitors.	Fellows.	Total.
Use of Library	12,945	1,558	14,503

The Library has been kept open two evenings each week and on the five minor legal holidays for the same period of time as during the previous year:

Evenings.	Visitors.	Fellows.	Total.
74	1,021	130	1,151
Legal holidays.			
5	86	28	114

These figures are included in the total given for the year:

	1932.	1931.	Increase.
Number of volumes consulted in the Library	85,102	63,296	21,806

The number of books "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and works of reference kept on the shelves in the Reading Room, and the Fellows of the College have access to the Book Stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

	1932.	1931.	Increase.
Number of volumes taken out	5,523	4,480	1,043

Use of Study Rooms: Average number of rooms in use throughout the year, 3.

Number of volumes in use in these rooms during the year, 1351.

	Works.	Volumes.	Cards written.	Printed cards revised.
Cataloguing	534	572	8,607	1,624

All books added to the shelves during the past year and 204 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 15,457 cards have been revised, rewritten and filed.

2835 unbound pamphlets and reprints have been subject-headed, and arranged alphabetically by subject and by author under the subject.

We have listed November 1, 1932, 1377 current periodical publications, including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Funds	130	268
By purchase from General Account	88	177
In exchange	97	139
Editors	95	25
Publishers	135	42
Gifts from various Donors	48	15
	593	666
Reports not included in the above	78	40
	671	706

The actual number of periodicals received during the year, including new subscriptions (3 American, 1 foreign), total 1259.

1888 dissertations and 288 theses have been added to our collections, from the following list of European schools of medicine with which we exchange publications at the present time:

University of	Amsterdam	University of	Lausanne
"	Basel	"	Leiden
"	Berlin	"	Leipzig
"	Bonn	"	Lund
"	Breslau	"	Marburg
"	Erlangen	"	Paris
"	Giessen	"	Rostock
"	Göttingen	"	Uppsala
"	Heidelberg	"	Utrecht
"	Kiel	"	Würzburg
"	Königsberg		

We have received in cash from the sale of duplicates for the current year, $54.

We have distributed books and journals on exchange account to the following:

Boston Medical Library, Boston, Mass., the School of Medicine, of the University of Pennsylvania, and, in addition, about sixty duplicates, by Philadelphian authors, were donated to the Library of American Authors, in Honor of Pasteur, University of Strasbourg, Strasbourg, France.

We have received exchanges from:

Houston Academy of Medicine, Houston, Texas
New York Academy of Medicine, New York City

By exchanges and purchases from the Funds, we have been able to complete the files of the following journals:

American Journal of Roentgenology
Anales de Oftalmologia
Anatomischer Bericht
Annali di Medicina Navale
Archiv für Thierheilkunde
Archiv für Wissenschaftliche und Praktische Thierheilkunde
Archives de Biologie
Archivio di Ortopedia
Archivio di Scienze Biologiche
Folia Medica

Giornale Internazionale delle Scienze Mediche
Quarterly Review of Biology
Revue d'Ophthalmologie
Rivista Critica di Clinica Medica
Zeitschrift für Augenheilkunde
Zeitschrift für Fleisch und Milchhygiene

In response to requests sent out during the year we have received acceptances from 19 foreign and 34 American publishers and editors to send their periodical publications as issued, either as a gift or in exchange for the Transactions of the College.

Amount of fines for the current year ending, November 1, 1932, $31.

List of Incunabula, rare medical books, and works of special interest, received during the year. (These rare books have been acquired without in any way interfering with the purchase of current medical publications.)

INCUNABULA

[Total number of Incunabula at this date, 406]

Albertus Magnus. Paradisus animae sive de virtutibus libellus. Argentorati, Martinus Flach, 1493. [Hain No. 481.] Gesamtkatalog No. 706.

Fund for Rare Books.

Beroaldus, Philippus. 1453–1505 A.D. Heptalogus s. Libellus, quo septem sapientum sententiae discuntiuntur.*** Bononiae, Benedictus Hectoris, Bononiensis, 1498. [Hain No. 2974.] Gesamtkatalog No. 4138.

Fund for Rare Books.

Gerson, John. 1363–1429 A.D. De modo vivendi omnium fidelium.*** Daventriae, circa 1490. [Hain No. 7671.] Gothic letter, large capitals painted in blue and red.

Fund for Rare Books.

Plato. Opera. Prohemium Marsilij Ficini Florentini in libros Platonis ad Laurentium Medicum virum magnanimum. Venetiis, Bernardinus de Choris Cremonesis et Simon de Luere, 1491. [Hain No. 13963.]

In Exchange, Boston Medical Library.

Works of Special Interest

Académie de chirurgie. Opuscules de chirurgie, par M. Morand. 2 pt. in 1 v. Paris, Desprez, 1768–1772.

Richard H. Harte Fund.

Barthez, Paul Joseph. Mémoires sur le traitement méthodique des fluxions, et sur les coliques iliaques qui sont essentiellement nerveuses. Montpellier, Sevalle, 1816.

Fund for Rare Books.

History of the British Medical Association. 1832–1932. Compiled by E. M. Little. London, 1932.

Presented by the Association.

Cooke, James. Mellificium chirurgie; or, The marrow of chirurgery much enlarged, to which is now added anatomy, illustrated with twelve brass cuts, and also the marrow of physick; both in the newest way. London, Shirley, 1676.

Purchased from Mr. O. V. Weaver, great-grandson of Joseph Priestley. Formerly the property of Lloyd Zachary, of Thomas Stacy and of John Derr Weaver, M.D., Univ. of Penna., 1861.

Dalton, John. Extraordinary facts relating to the vision of colors; with observations. In Memoirs of the Literary and Philosophical Society of Manchester. 1798. v. 5, pt. 1, pp. 28–29.

Fund for Rare Books.

Emmons, Samuel B. Vegetable family physician; containing a description of the roots and herbs common to this country, with their medicinal properties and uses; also directions for the treatment of the diseases incident to human nature, by vegetables alone; embracing many valuable Indian recipes. Boston, Oakes, 1836.

Douglas Stockton Warren Memorial Fund.

Fabricius ab Aquapendente, Hieronymus. Opera chirurgica. Quorum pars prior pentateuchum chirurgicum, posterior operationes chirurgicas continet.*** Patavii, imp. Francisci Rolzettae, 1647.

Fund for Rare Books.

Faculté de médecine de Paris. Statuta facultatis medicinae parisiensis. Parisiis, apud Franciscum Muguet, 1696.

Fund for Rare Books.

Galen, Claudius. Methodus medendi, vel de morbis curandi libri quatuordecim. Thoma Linacro Anglo interprete. Parisiis, apud Simonem Colinaeum, 1530.

Fund for Rare Books.

Harper, Lathrop C. A selection of incunabula describing one thousand books printed in the fifteenth century. New York. From the stock of Lathrop C. Harper, 1920.

Presented by the Author.

Hunain Ibn Is-Haq. The book of the ten treatises on the eye ascribed to Hunain Ibn Is-Haq [809–877 A.D.]. The earliest existing systematic textbook of ophthalmology. The Arabic text edited from the only two known manuscripts, with an English translation and glossary by Max Meyerhof, oculist in Cairo [Egypt]. Cairo, Government Press, 1928.

S. Lewis Ziegler Fund.

Jeanselme, Edouard. Histoire de la syphilis.*** Paris, Doin, 1931. Extr.-Traité de la syphilis. 1931. v. 1.

Presented by E. Jeanselme, M.D.

Lambert, Antoine. Commentaires ov les oevvres chirurgicales.*** 2 ed. Lyon, Campagnon, 1671.

Fund for Rare Books.

[de la Mettrie, Julien Offray.] Man a machine. Translated from the French of the Marquiss d'Argens. [Pseudonym.] London, Owen, 1749.

Fund for Rare Books.

LeCat, Claude Nicolas. Pièces concernant l'operation de la taille. 2 v. Rouen, du Mesnil, 1746.

Richard H. Harte Fund.

Manoel. [Emanuel II, King of Portugal.] Early Portuguese books, 1489–1600, in the library of His Majesty, King of Portugal. 2 v. London, Maggs, 1929–1932.

Fund for Rare Books.

Mueller, Joh. De respiratione foetus; commentatio physiologica, in Academia borussica rhenana praemio ornata. Lipsiae, apud Carolum Cnoblochium, 1823.

Fund for Rare Books.

Munk, William. Life of Sir Henry Halford, Bart. Physician to George III, George IV, William IV and Queen Victoria. London, Longmans, 1895. Extra-illustrated. Forty plates inserted.

Fund for Rare Books.

Sabattini, Giambattista. Tavole anatomiche per li pittori, e gli scultori. Bologna, tip. del Fratelli Masi, e comp., 1814.

Fund for Rare Books.

Smith, Sir Frederick. Early history of veterinary literature and its British development. London, Baillière, 1924–1930.

Presented by Baillière, Tindall & Cox.

Stillwell, Margaret Bingham. Incunabula and americana, 1450–1809. A key to bibliographical study. New York, Columbia University Press, 1931.

John D. Griscom Fund.

Sylvius de la Boë, Franciscus. Opera medica,*** quibus hac in editione accesserunt Causus medicinales, annor. M.D.C.LIX.LX & LXI. Genevæ, apud Fratres de Tournes, 1698.

Fund for Rare Books.

Manuscripts

Wood, George B. Notes of lectures on medical practice, taken by J. Solis-Cohen.

Presented by Myer Solis-Cohen, M.D.

Owen, Sir Richard. Original manuscripts on "Trichina Spiralis."

Presented by E. B. Krumbhaar, M.D.

Snodgrass, L. E.: Life and works of G. G. Davis, M.D. [Typewritten.]

Presented by the Author.

Snodgrass, L. E. Diagrams of braces devised by G. G. Davis, M.D.

Presented by the Author.

Hill, A. V.: Present tendencies and methods of physiological teaching and research.

Presented by E. B. Krumbhaar, M.D.

Other Interesting Additions.

Bronze Plaque of Francis Xavier Dercum, M.D. [Made in 1924 by Carl August Heber, Sculptor.]

Presented by Mrs. F. X. Dercum.

We have received during the past year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 199 prints and to other than Fellows, on application, 1176 prints.

The work for the Library has been continued as heretofore; 1842 large and small prints have been made, including 6 volumes of Incunabula and various portraits and manuscripts.

Up to the present time there have been made photostat reproductions of 299 Incunabula.

A complete list of all the Funds and the expenditures for the year is given in a previous part of this report. All additional

expenses, salaries, binding, etc., have to be governed according to the funds annually appropriated by the College to the Library Committee.

An account of stock has been taken and any displaced books found arranged in their proper order, and replacements made.

Mr. Charles Perry Fisher, Librarian, having served in this capacity for fifty years, retired, July 1, 1932, and Dr. Emlen Wood was appointed Acting Librarian.

Library Committee:
Albert P. Brubaker, M.D.,
Chairman.

INDEX

A

Adler, F. H., discussion of case presenting disjunctive ocular movements, 268
on case of bullous keratitis successfully treated by iridectomy, 271
on diagnosis of double perforation of globe, 248
on pulsating exophthalmos, 262
Animals, cancer in, 152
Appendicitis, increasing incidence and morbidity of, 76
Appendix, introductory note, 230
Appleman, Leighton F., case of pulsating exophthalmos, 262
discussion on posterior lenticonus, 233
report upon 100 extracapsular cataract extractions by Knapp method, 272, 275
Ashhurst, Astley P. C., and Klopp, John W., prognosis in carcinoma of stomach, remote results of surgical treatment to January, 1930, 116

B

Babbitt, James A., discussion on ear, nose and throat clinic, 303
on present-day theories on etiology and pathology of nasal-sinus infection, 325
on sinusitis in children, 292
pulmonary complications of tonsillectomy, with report of three cases, 320, 323
Batson, Oscar V., discussion on otological problem presented by suppuration in petrous pyramid, 316
Bond, Earl D., experiment in psychoanalysis, 69
Bower, John O., increasing incidence and morbidity of appendicitis, 76
Bradbury, Samuel, plan of British Medical Association for general medical service for nation, 44
Bricker, Samuel, discussion on otological problem presented by suppuration in petrous pyramid, 317
British Medical Association, plan of, for general medical service for nation, 44
Brown, Lawrason, what Koch meant to tuberculosis, 211
Buchanan, Mary, two cases of retained pupillary membranes, 261

C

Cancer, as a problem of biology of cell division, 136
in animals, 152
in man, 112
summarization of advance in our knowledge of some features of, 193

Carcinoma of stomach, prognosis in, remote results of surgical treatment to January, 1930, 116
Carpenter, Howard C., and Stokes, Joseph, Jr., preliminary report concerning prophylactic use of parent's blood during anterior poliomyelitis epidemic of 1932, 171
Cell division, biology of, cancer, as problem of, 136
Chance, Burton, discussion concerning superficial punctate keratitis and associated conjunctivitis, 279
on Leber's disease, 259
on venereal conjunctivitis, 283
extraction within capsule of crystalline lens containing foreign body, 277
Goethe and his theory of color, 225
intubation of cases, 255
sketch of Dr. Burton Alexander Randall as an ophthalmologist, 266
Clarke, Alexander, discussion on vernal conjunctivitis, 253
Clerf, Louis H., discussion on pulmonary complications of tonsillectomy, 322
on some experience in treatment of bilateral abductor paralysis, 312
Clinic, tumor, 145
Coates, George M., discussion on pulmonary complications of tonsillectomy, 322
of some recent developments in regard to otosclerosis, cholesteatoma and static labyrinth, 333
on studies of pathologic temporal bones in children, 329
Corneal dystrophy, 231
Cornell, Walter S., review of some recent medical literature relating to medical economics, costs of illness, and organization of medical practice, 58
Costs on medical care, purposes of committee on, 37
Cowan, Alfred, discussion on corneal dystrophy, 232
on diagnosis of double perforation of globe, 247
on posterior lenticonus, 234
Cross, G., discussion on intracapsular extraction by Knapp method, 274
on sarcoma of choroid, 236
on use of snare in enucleation of eyeball, 240

D

Davis, Warren B., sinusitis in children, 288, 292
Deformity in infantile paralysis, prevention of, 33
De Long, Perce, discussion on inclusion operation, 237
metastatic carcinoma of choroid, 280
new technique for preparation of pathological specimens for microscopic study, 273

E

Ersner, Matthew S., discussion on bronchial asthma, 299
on ear, nose and throat clinic, 303
on otological problem presented by suppuration of petrous pyramid, 317
on prophylaxis and treatment of septic meningitis, 307
on pulmonary complications of tonsillectomy, 322
Eves, Curtis C., discussion on present-day theories concerning etiology and pathology of nasal sinus infection, 325

F

Facial expression of emotions, with special reference to violent effort and fatigue, 174
Faulkner, E. Ross (New York), discussion on atresia of choanæ, its incidence types and cause, 333
Fellows, Haynes Harold, incidence of tuberculosis in industrial workers as revealed by fluoroscopic examination, 90
Fewell, A. G., unilateral neuroretinitis of luetic origin with stellate figure at macula, 238
Flexner, Simon, poliomyelitis, mode of infection and means of prevention, 11
Fox, Herbert, discussion on cancer in animals, 156
Fry, W. E., discussion on new technique for preparation of pathological specimens, 272
metastatic sarcoma of choroid, 243

G

Gibbon, John H., importance of differential diagnosis of tumors, 139
Gittings, J. C., incidence of tuberculosis in infants and pre-school children and conditions under which they become infected, 84
Gleason, Edward B., discussion on otological problem presented by suppuration in petrous pyramids, 317
Goethe and his theory of color, 225
Grant, Francis C., discussion on otological problem presented by suppuration in petrous pyramid, 316
Griscom, J. Milton, a corneal dystrophy, 231

H

Hammett, Frederick S., cancer as a problem of biology of cell division, 136
Hauser, Karl M., discussion on bronchial asthma, 299
Henry, J. Norman, general remarks on outbreak of poliomyelitis in Philadelphia, 163
Herman, Maxwell, discussion on amaurosis, 265
Hetherington, H. W., discussion on tuberculous lesions, 107
Hewson, William, discussion on prophylaxis and treatment of septic meningitis, 308
intimate notes on ear, nose and throat clinic, 300, 303
Holloway, T. B., discussion concerning superficial punctate keratitis and associated conjunctivitis, 279
on corneal dystrophy, 234
on posterior lenticonus, 234
on unilateral neuroretinitis of luetic origin, 238
on vernal conjunctivitis, 254, 284
Leber's disease, 258

Hoover, W. B. (by invitation) some experience in treatment of bilateral abductor paralysis, 308, 313
Houser, Karl M., discussion on sinusitis in children, 292
on pulmonary complications of tonsillectomy, 322

I

Infantile paralysis, prevention of deformity in, 33
Insurance, social, and health work in Germany and Austria, 41

J

Jackson, Chevalier, postoperative pulmonary complications, are they preventable? 330, 331
Jackson, Chevalier L., preventriculosis (so-called cardiospasm) diagnosis and treatment (moving picture film demonstration), 338

K

Kern, Richard A., discussion on bronchial asthma, 298
Kernan, J. D., discussion on postoperative pulmonary complications, are they preventable? 331
Klauder, Joseph V., syphilis in the third generation, 260
Kolmer, John A., and Amano, K. (by invitation), studies on prophylaxis and treatment of septic meningitis, 305
Kolmer, John A., discussion on bronchial asthma, 298
Kopetsky, Samuel J. (by invitation), otological problem presented by suppuration in petrous pyramid, 313, 317

L

Langdon, H. Maxwell, and (by invitation) James E. Aiguier, fracture of maxilla through left optic foramen, reduction with Kingsley splint, with restoration of vision, 276, 277
and Laws, G. M., amaurosis after uterine hemorrhage, with restoration of vision following transfusion, 262, 265, 266
Langdon, H. Maxwell, discussion on Leber's disease, 259
on case of bullous keratitis successfully treated by iridectomy, 271
of case presenting disjunctive ocular movements, 269
on diagnosis of double perforation of globe, 248
on intracapsular extraction by Knapp method, 274
on metastatic carcinoma of choroid, 282
intraocular sarcoma, 245
melanotic sarcoma of choroid occurring at optic disk in eye with previous detachment of retina, 256
wound of globe by explosion of shatter-proof wind shield (Presentation of case), 255

Lehrfeld, Louis (by invitation), observations on eighty-seven cases of vernal conjunctivitis at Wills hospital, 250, 254
sodium carbonate in treatment of vernal conjunctivitis, 282
Lewis, Fielding O., total laryngectomy, one-stage operation (lantern-slide demonstration of various phases and steps in this operation), 341
Library Committee, Annual Report for 1932, 349
Literature, some recent medical, relating to economics, costs of illness and organization of medical practice, 58

M

Man, cancer in, 112
McKenzie, R. Tait, M.D., LL.D., R.C.A., facial expression of emotions, with special reference to violent effort and fatigue, 174
McNally, William J. (by invitation), nature of static labyrinth as determined by experiments in amphibia, 318
McPhedran, F. Maurice, occurrence and progress of tuberculous lesions in school children and adults, 98
Medical care, purposes of committee on costs of, 37
Moore, William, discussion on pulmonary complications of tonsillectomy, 322
Morgan, Arthur C., purposes of committee on costs of medical care, 37

N

Nye, Robert B., Curtis Clinic, its purpose and possible influence for advancement of community health, 53

O

Ophthalmology, proceedings of section on, 231
Opie, Eugene L., exogenous tuberculous infection of adults, 108

P

Packard, Francis R., president's address, 1
Paralysis, infantile, prevention of deformity, 33
Parish, Benjamin D., discussion on pulmonary complications of tonsillectomy, 322
present-day theories concerning etiology and pathology of nasal sinus infections, 323, 325
Paul, John R., and (by invitation) Trask, J. D., observations on epidemiology of poliomyelitis, 158
Pendergrass, E. P., discussion of incidence of tuberculosis, 96
Peter, Luther C., discussion on report of 100 intracapsular extractions by Knapp method, 273
enucleation of eyeball, 239, 241
on metastatic sarcoma of choroid, 245

Pfahler, G. E., lymphatic drainage from maxillary sinuses, 293, 294
Poliomyelitis epidemic of 1932, preliminary report concerning prophylactic use of parent's blood during, 171
epidemiology of, observations on, 158
mode of infection and means of prevention, 11
outbreak in Philadelphia, general remarks on, 163
Posey, W. C., discussion on Leber's disease, 260
President's address, 1
Proceedings of Section on Medical History, 346
on General Medicine, 342
on Otology and Laryngology, 285
on Public Health and Industrial Medicine, 345
Psychoanalysis, experiment in, 69

R

Ratcliffe, Herbert L., Sc. D., cancer in animals, 152
Reese, Warren S., discussion on diagnosis of double perforation of globe, 247
exhibition of case presenting disjunctive ocular movements, 268
posterior lenticonus, 232
Reiman, Stanley P., cancer in man, 112
Ridpath, Robert F., actinomycosis of tongue and tonsil, 304
Roberts, Walter, and Dintenfass, Henry, operation on abscess of temporo-sphenoidal lobe (moving picture film demonstration), 340

S

Scarlett, Hunter W., discussion concerning superficial punctate keratitis and associated conjunctivitis, 279
microphthalmos with coloboma of iris, choroid and disk, 257
Schaeffer, J. Parsons, anatomy of paranasal sinuses in children, 285
atresia of choanæ, its incidence, types and cause, 331
discussion on lymphatic drainage from maxillary sinuses, 294
Schatz, H. A., discussion on sinusitis in children, 292
Schenck, Harry P., bronchial asthma from rhinological standpoint, 296
deSchweinitz, G. E., Baer, B. F., Jr., and DeLong, Perce, sarcoma of choroid, 235
discussion on amaurosis, 265
on case of primary sarcoma of iris, 270
on metastatic carcinoma of choroid, 243, 244
on vernal conjunctivitis, 254
remarks concerning superficial punctate keratitis and associated conjunctivitis, 277
Shannon, C. E. G., discussion on intracapsular extraction by Knapp method, 274
on Leber's disease, 260

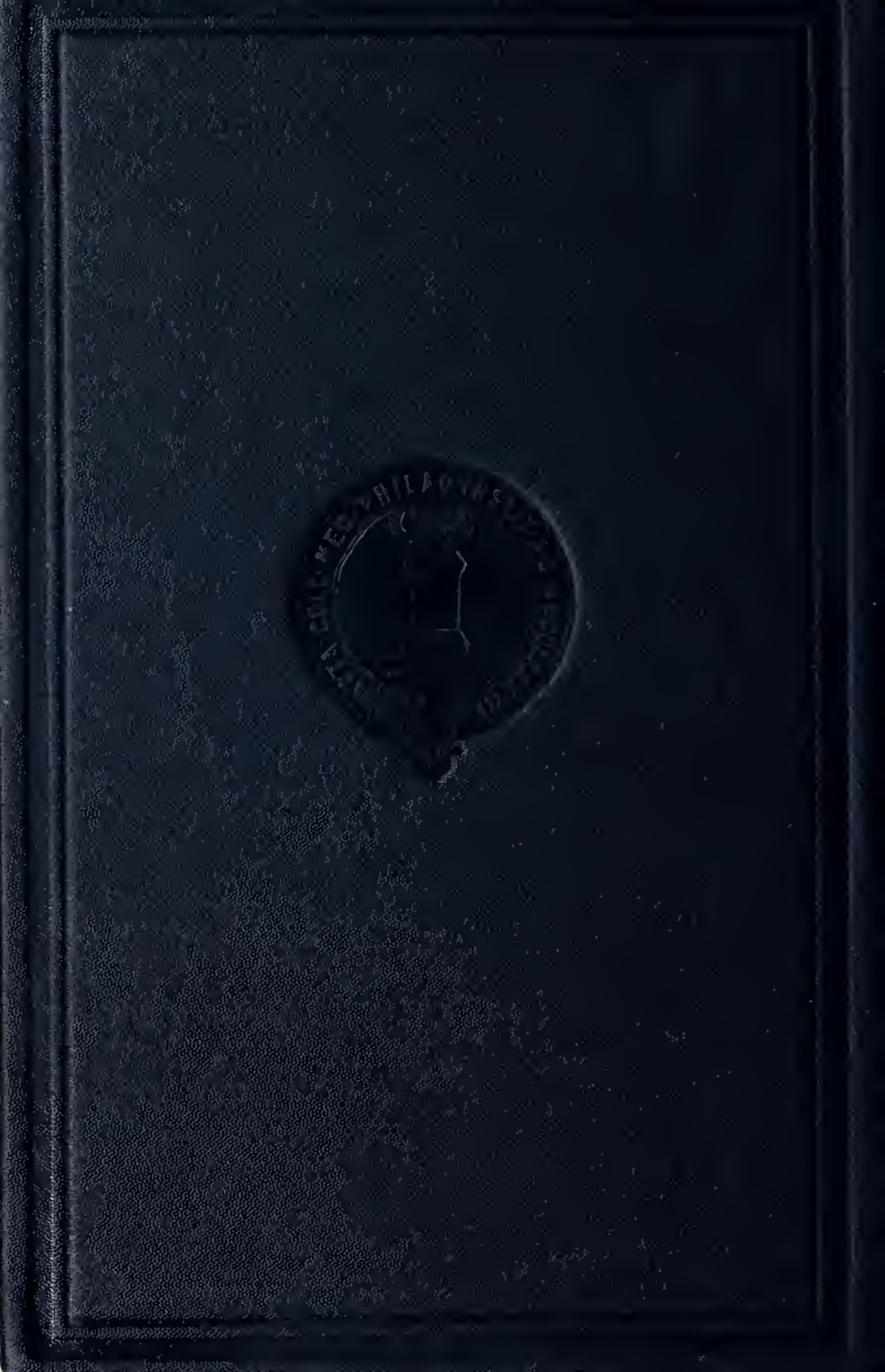
Shumway, E. A., discussion concerning superficial punctate keratitis and associated conjunctivitis, 279
on amaurosis, 264
on fracture of maxilla through left optic foramen, 276
on metastatic carcinoma of choroid, 282
on vernal conjunctivitis, 254, 283
Shuster, H. H., discussion on nature of static labyrinth, 319
Simpson, Burton T., tumor clinic, 145
Skillern, S. R., discussion on anatomy of paranasal sinuses in children, 288
Skillern, Samuel R., Jr., discussion on bronchial asthma, 299
on prophylaxis and treatment of septic meningitis, 308
Smyth, Henry Field, on social insurance and health work in Germany and Austria, 41
Spackman, E. W., diagnosis of double perforation of globe by injection of air into space of Tenon, 246, 249
Spaeth, E. B., discussion on use of snare in enucleation of eyeball, 240
inclusion operation on rabbit, 237
Spiller, William G., discussion of case presenting disjunctive ocular movements, 268
Stokes, Joseph, Jr., discussion on tuberculosis, 89
Stout, Philip, discussion on ear, nose and throat clinic, 303
on lymphatic drainage from maxillary sinuses, 294
on present-day theories concerning etiology and pathology of nasal-sinus infections, 325
on prophylaxis and treatment of septic meningitis, 307
on pulmonary complications of tonsillectomy, 323
on sinusitis in children, 292
on studies on pathologic temporal bones in children, 329
Strecker, Edward A., discussion on psychoanalysis, 74

T

Teller, Miss Ida (by invitation), allergic investigations on twenty-three cases of vernal conjunctivitis, 249
Tuberculosis, exogenous, infection of adults, 108
incidence of infants and pre-school children and conditions under which they become infected, 84
in industrial workers as revealed by fluoroscopic examination, 90
Tuberculosis, what Koch meant to, 211
Tuberculous lesions, occurrence and progress of, in school children and adults, 98
Tucker, Gabriel, discussion on bronchial asthma, 299
on pulmonary complications of tonsillectomy, 320, 323
on some experience in treatment of bilateral abductor paralysis, 312
laryngofissure for early laryngeal cancer (moving picture film demonstration), 339
Tumor clinic, 145
Tumors, differential diagnosis of, importance of, 139

W

Wagers, Arthur, discussion on bronchial asthma, 298
Waugh, Justin M., discussion on bronchial asthma, 299
Wells, H. Gideon, summarization of advance in our knowledge of some features of cancer, 193
Widmann, B. F., discussion on vernal conjunctivitis, 251
Willard, De Forest P., prevention of deformity in infantile paralysis, 33
Wilmer, H. B., sinusitis and rôle it plays in production of asthma, 294, 299
Wolff, Robert M., studies on pathologic temporal bones in children, 326, 329
Wood, George, discussion on bronchial asthma, 299
on pulmonary complication of tonsillectomy, 322

Z

Zentmayer, William, case of bullous keratitis successfully treated by iridectomy, 270
of bilateral metastatic carcinoma of choroid, 241
of primary sarcoma of iris, 269, 270
discussion on amaurosis, 264
on diagnosis of double perforation of globe, 248
on Leber's disease, 258
on pulsating carotid artery, 262
on unilateral neuroretinitis of luetic origin, 239
on use of snare in enucleation of eyeball, 240